

- [33] H. Christensen, J. Schüz, M. Kosteljanetz, H. Poulsen, J. Thomsen, C. Johansen, Cellular telephone use and risk of acoustic neuroma, *Am. J. Epidemiol.* 159 (2004) 277–283.
- [34] M. Schoemaker, A. Swerdlow, A. Ahlbom, A. Auvinen, K. Blaasaas, E. Cardis, et al., Mobile phone use and risk of acoustic neuroma: results of the Interphone case-control study in five North European countries, *Br. J. Cancer* 93 (2005) 842–848.
- [35] T. Takebayashi, S. Akiba, Y. Kikuchi, M. Taki, K. Wake, S. Watanabe, et al., Mobile phone use and acoustic neuroma risk in Japan, *Occup. Environ. Med.* 63 (2006) 802–807.
- [36] B. Schlehofer, K. Schlaefter, M. Blettner, G. Berg, E. Böhler, I. Hettinger, et al., Environmental risk factors for sporadic acoustic neuroma (Interphone Study Group, Germany), *Eur. J. Cancer* 43 (2007) 1741–1747.
- [37] L. Hardell, K. Hansson Mild, M. Carlberg, A. Hallquist, Cellular and cordless telephones and the association with brain tumours in different age group, *Arch. Environ. Health* 59 (3) (2004) 132–137.
- [38] L. Hardell, K. Hansson Mild, M. Carlberg, Use of cellular telephones and brain tumour risk in urban and rural areas, *Occup. Environ. Med.* 62 (2005) 390–394.
- [39] L. Hardell, A. Hallquist, K. Hansson Mild, M. Carlberg, H. Gertzén, E. Schildt, et al., No association between the use of cellular or cordless telephones and salivary gland tumours, *Occup. Environ. Med.* 61 (8) (2004) 675–679.
- [40] S. Lönn, A. Ahlbom, H. Christensen, C. Johansen, J. Schüz, S. Edström, et al., Mobile phone use and risk of parotid gland tumor, *Am. J. Epidemiol.* 164 (7) (2006) 637–643.
- [41] S. Sadezki, A. Chetrit, A. Jarus-Hakak, E. Cardis, Y. Deutch, S. Duvdevani, et al., Cellular phone use and risk of benign and malignant parotid gland tumors—a nationwide case-control study, *Am. J. Epidemiol.* 167 (4) (2008) 457–467.
- [42] L. Hardell, M. Eriksson, Is the decline of the increasing incidence of non-Hodgkin lymphoma in Sweden and other countries a results of cancer preventive measures? *Environ. Health Perspect.* 111 (2003) 1704–1706.
- [43] L. Hardell, Pesticides, soft-tissue sarcoma and non-Hodgkin lymphoma—historical aspects on the precautionary principle in cancer prevention, *Acta Oncol.* 47 (3) (2008) 347–354.
- [44] M. Eriksson, L. Hardell, M. Carlberg, M. Åkerman, Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis, *Int. J. Cancer* 123 (2008) 1657–1663.
- [45] L. Hardell, M. Eriksson, M. Carlberg, C. Sundström, K. Hansson Mild, Use of cellular or cordless telephones and the risk for non-Hodgkin's lymphoma, *Int. Arch. Occup. Environ. Health* 78 (8) (2005) 625–632.
- [46] M.S. Linet, T. Taggart, R.K. Severson, J.R. Cerhan, W. Cozen, et al., Cellular telephones and non-Hodgkin lymphoma, *Int. J. Cancer* 119 (2006) 2382–2388.
- [47] L. Hardell, B. van Bavel, G. Lindström, M. Carlberg, A.C. Dreifaldt, H. Wijkström, et al., Increased concentrations of polychlorinated biphenyls, hexachlorobenzene and chlordanes in mothers to men with testicular cancer, *Environ. Health Perspect.* 111 (2003) 930–934.
- [48] L. Hardell, B. van Bavel, G. Lindström, M. Eriksson, M. Carlberg, In utero exposure to persistent organic pollutants in relation to testicular cancer, *Int. J. Androl.* 29 (2006) 228–234.
- [49] L. Hardell, N. Malmqvist, C.G. Ohlson, H. Westberg, M. Eriksson, Testicular cancer and occupational exposure to polyvinyl chloride plastics: a case-control study, *Int. J. Cancer* 109 (2004) 425–429.
- [50] L. Hardell, M. Carlberg, C.G. Ohlson, H. Westberg, M. Eriksson, K. Hansson Mild, Use of cellular and cordless telephones and risk of testicular cancer, *Int. J. Androl.* 30 (2) (2007) 115–122.
- [51] A. Stang, G. Anastassiou, W. Ahrens, K. Broman, N. Bornfeld, K. Jöckel, The possible role of radiofrequency radiation in the development of uveal melanoma, *Epidemiology* 12 (2001) 7–12.
- [52] C. Johansen, J.D. Boice Jr., J.K. McLaughlin, H.C. Christensen, J.H. Olsen, Mobile phones and malignant melanoma of the eye, *Br. J. Cancer* 86 (2002) 348–348.
- [53] P.D. Inskip, Frequent radiation exposures and frequency dependent effects: the eyes have it. (Editorials), *Epidemiology* 12 (2001) 1–4.
- [54] H. Warren, A. Prevatt, K. Daly, P. Antonelli, Cellular telephone use and risk of intratemporal facial nerve tumor, *Laryngoscope* 113 (2003) 663–667.
- [55] L. Hardell, M. Carlberg, K. Hansson Mild, Methodological aspects of epidemiological studies on the use of mobile phones and their association with brain tumors, *Open Environ. Sci.* 2 (2008) 54–61.
- [56] S. Lönn, Mobile phone use and risk of intracranial tumors, Medical Dissertation, Karolinska Institute, Stockholm, Sweden, 2004.
- [57] H.O. Adami, A. Ahlbom, A. Ekblom, L. Hagmar, M. Ingelman-Sundberg, Opinion—“Experts who talk rubbish”, *Bioelectromag. Soc. Newslett.* 162 (2001) 4–5.
- [58] L. Hardell, M. Walker, B. Walhjalt, L.S. Friedman, E.D. Richter, Secret ties to industry and conflicting interests in cancer research, *Am. J. Ind. Med.* 50 (2007) 227–233.
- [59] Risk evaluation of potential environmental hazards from low frequency electromagnetic field exposure using sensitive *in vitro* methods. Final Report, Available from: <[http://www.itis.ethz.ch/downloads/REFLEX\\_Final%20Report\\_171104.pdf](http://www.itis.ethz.ch/downloads/REFLEX_Final%20Report_171104.pdf)>.
- [60] D. Trichopoulos, H.O. Adami, Cellular telephones and brain tumors, *N. Engl. J. Med.* 344 (2) (2001) 133–134.
- [61] D. Michaels, Doubt is Their Product. How Industry's Assault on Science Threatens Your Health, Oxford University Press, New York, 2008.
- [62] K. Hansson Mild, L. Hardell, M. Kundi, M.O. Mattsson, Mobile phones and cancer: is there really no evidence of an association? (Review), *Int. J. Mol. Med.* 12 (2003) 67–72.
- [63] M. Vrijheid, E. Cardis, B. Armstrong, A. Auvinen, G. Berg, K. Blaasaas, et al., Validation of short term recall of mobile phone use for the Interphone study, *Occup. Environ. Med.* 63 (4) (2006) 237–243.
- [64] M. Vrijheid, I. Deltour, D. Krewski, M. Sanchez, E. Cardis, The effects of recall errors and of selection bias in epidemiologic studies of mobile phone use and cancer risk, *J. Expo. Sci. Environ. Epidemiol.* 16 (4) (2006) 371–384.
- [65] M. Vrijheid, L. Richardson, B.K. Armstrong, A. Auvinen, G. Berg, M. Carrol, et al., Quantifying the impact of selection bias caused by non-participation in a case-control study of mobile phone use, *Annals Epidemiol.* 19 (2009) 33–41.



ELSEVIER

Pathophysiology xxx (2009) xxx–xxx

ISP  
PATHOPHYSIOLOGY

www.elsevier.com/locate/pathophys

# Mobile phone base stations—Effects on wellbeing and health

Michael Kundi\*, Hans-Peter Hutter

*Institute of Environmental Health, Center for Public Health, Medical University of Vienna,  
Kinderspitalgasse 15, A-1095 Vienna, Austria*

Received 11 September 2008; accepted 30 January 2009

## Abstract

Studying effects of mobile phone base station signals on health have been discouraged by authoritative bodies like WHO International EMF Project and COST 281. WHO recommended studies around base stations in 2003 but again stated in 2006 that studies on cancer in relation to base station exposure are of low priority. As a result only few investigations of effects of base station exposure on health and wellbeing exist. Cross-sectional investigations of subjective health as a function of distance or measured field strength, despite differences in methods and robustness of study design, found indications for an effect of exposure that is likely independent of concerns and attributions. Experimental studies applying short-term exposure to base station signals gave various results, but there is weak evidence that UMTS and to a lesser degree GSM signals reduce wellbeing in persons that report to be sensitive to such exposures. Two ecological studies of cancer in the vicinity of base stations report both a strong increase of incidence within a radius of 350 and 400 m respectively. Due to the limitations inherent in this design no firm conclusions can be drawn, but the results underline the urgent need for a comprehensive investigation of this issue. Animal and in vitro studies are inconclusive to date. An increased incidence of DMBA induced mammary tumors in rats at a SAR of 1.4 W/kg in one experiment could not be replicated in a second trial. Indications of oxidative stress after low-level in vivo exposure of rats could not be supported by in vitro studies of human fibroblasts and glioblastoma cells.

From available evidence it is impossible to delineate a threshold below which no effect occurs, however, given the fact that studies reporting low exposure were invariably negative it is suggested that power densities around 0.5–1 mW/m<sup>2</sup> must be exceeded in order to observe an effect. The meager data base must be extended in the coming years. The difficulties of investigating long-term effects of base station exposure have been exaggerated, considering that base station and handset exposure have almost nothing in common both needs to be studied independently. It cannot be accepted that studying base stations is postponed until there is firm evidence for mobile phones.

© 2009 Published by Elsevier Ireland Ltd.

**Keywords:** Mobile phone base station; Performance; Cancer; In vitro studies; Microwaves

## 1. Introduction

Modern mobile telecommunication is based on a cellular system. Each cell is covered by a base station that keeps track of the mobile phones within its range, connects them to the telephone network and handles carry-over to the next base station if a customer is leaving the coverage area. Early mobile telecommunication systems had very large cells with tens of kilometers radius and were predominantly located along highways due to offering service mainly for car-phones. With the introduction of digital mobile phone systems cell sizes got much smaller and base stations were erected in densely

populated areas. The limited power of mobile phones made it necessary to reduce the distance to the customers. The cell size depends on (1) the radiation distance of the mobile phone; (2) the average number of connected calls; (3) the topographic characteristics of the covered area and the surrounding buildings, vegetation and other shielding objects; and (4) the type of antenna used. There are essentially three types of cells presently making up mobile telecommunication networks: (1) macro-cells in areas of average to low number of calls; (2) micro-cells in densely populated areas and areas with high telecommunication traffic density; (3) pico-cells within buildings, garages, etc. The types of antennas used, although hundreds of different models are operated, can be subdivided into: omni-directional antennas that radiate in all horizontal directions with the same power; sector antennas

\* Corresponding author. Tel.: +43 1 4277 64726; fax: +43 1 4277 9647.  
E-mail address: michael.kundi@meduniwien.ac.at (M. Kundi).

that radiate the main beam in one sector only but have varying aperture (usually 120° or 90°). These antennas can be mounted on masts (that sometimes are in the shape of trees for protection of landscape or are otherwise hidden), on the top of buildings, on pylons, and micro- and pico-cell antennas on various other places (walls of houses, shops, indoors, etc.). The width of the beam in vertical direction is typically 6°, but due to the presence of side lobes the actual pattern is more complicated.

Digital base stations of the second generation (GSM, TDMA) and third generation (UMTS, CDMA) have typically a nominal power for each channel of 10–20 W, micro- and pico-cells up to about 4 and 2 W, respectively. Due to the antenna gain the EIRP in the direction of the main beam is much greater (by a factor of  $10^{g/10}$ , where  $g$  is the antenna gain in dB, typically between 40 and 60). Most base stations of the second generation operate with two channels, one broadcast control channel (BCCH, channel used for transmitting information about the network, the location area code, frequencies of neighboring cells, etc.) and one traffic channel (TCH, channel used for transmission of calls), for third generation systems, due to code division multiplexing, control information needed for the maintenance of the system is at present transmitted together with the actual information (calls, pictures, etc.) within one broad-band channel. GSM systems operate the BCCH with all time slots occupied and therefore at maximal power, whereas TCH has as many time slots active as necessary to operate all active transmission not covered by the BCCH. Field strength at ground level depends on the characteristics of the antenna. Because the main beam reaches ground level typically in 50–200 m distance, in case of free sight to the antenna, maximum field strength is reached at that distance. However, due to the side lobes ups and downs of field strength occur as one approach the base station. In areas where objects are shadowing the beams, patterns are still more complex because of diffraction and reflection and multi-path propagation with constructive as well as destructive interference.

Free field propagation from the antenna along the main beam follows the law:  $P(x) = \text{EIRP}/(4\pi \cdot x^2)$ , with  $P(x)$  the power flux density in  $x$  meters distance and EIRP the equivalent isotropic radiated power of the antenna. Significant deviations from this expectation occur due to the side lobes, presence of interfering objects, differences in vertical beam width, and variations in the number of active transmissions. For these reasons distance to the antenna is a poor proxy for exposure level.

Since the early 1990s tens of thousands of base stations have been erected in countries where digital networks were introduced. While older systems with their low number of base stations have hardly received public attention, the vast increase in base stations has led to public concerns all over the world. Anecdotal reports about various effects on wellbeing and health have led also to an increased awareness of physicians [1,2] and increased research efforts have been demanded [3]. Despite these professional and public con-

cerns, the WHO International EMF Project has discouraged research into effects of base stations, because it deemed research into effects of mobile phones of higher priority. This position was changed in 2003 when the new research agenda recommended studies around base stations. In 2006 it was again stated that research into potential health effects of base station is of low priority [4].

Due to these circumstances only very few investigations of effects of base stations on wellbeing and health exist. In addition some experimental studies have been conducted, most of which address the problem of short-term effects on complaints and performance.

The following review summarizes available evidence and critically assesses the investigations as to their ability to support or dismiss a potential effect of microwave exposure from base stations on wellbeing and health.

## 2. Epidemiological investigations

### 2.1. Wellbeing and performance

Santini et al. [5,6] report results of a survey in France to which 530 individuals (270 men and 260 women) responded. Study subjects were enrolled through information given by press, radio, and website, about the existence of a study on people living near mobile phone base stations. Frequency for each of 18 symptoms was assessed on a 4 level scale (never, sometimes, often, and very often). Participants estimated distance to the base station using the following categories: <10 m, 10–50 m, 50–100 m, 100–200 m, 200–300 m, >300 m. For comparison of prevalence of symptoms >300 m served as reference category. For all symptoms a higher frequency of the categories ‘often’ or ‘very often’ was found at closer (self-reported) distance to the base station. Fatigue, headaches, and sleeping problems showed highest relative increase. Due to a less than optimal statistical analysis comparing each distance category separately with the reference category the overall response pattern can only be assessed qualitatively. Fig. 1 shows relative prevalence averaged over all symptoms as a function of self-reported distance to the antenna. Interestingly the function is not monotonous but shows, after an initial drop, an increase at a distance of 50–100 m. Because of the fact that in many cases this is the distance at which the main beam reaches ground level this may indicate a relationship to actual exposure levels.

This study was a first attempt to investigate a potential relationship between exposure to base station signals and health and has, therefore, several shortcomings: (1) participants selected themselves into the study group by responding to public announcements; (2) distance was self-reported and no attempt was made to validate these reports (a German cross-sectional study in over 30,000 households revealed that more than 40% did not know they were living in the vicinity of a base station [7]); (3) no assessment of subjects’ concerns about the base station; and (4) no measurement or calcula-

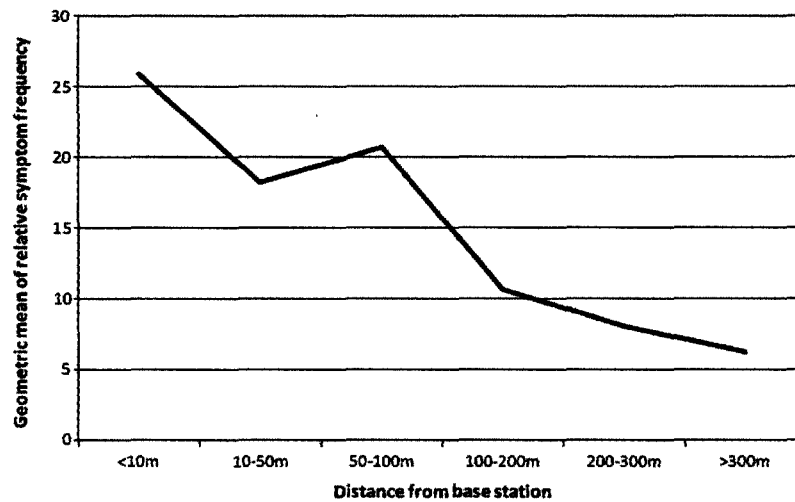


Fig. 1. Relative symptom frequency averaged over all 33 reported symptoms from Santini et al. [5] as a function of distance from base station.

tion of actual exposure. Although selection bias and wrong estimation of distance to the base station could have led to a spuriously increased prevalence of symptoms, the pattern of symptom frequency as a function of distance is intriguing and suggests that part of the increased symptom prevalence could be due to exposure because people do not know the typical pattern of field strengths found in the vicinity of base stations.

A Spanish version of the questionnaire as applied in the French study was distributed in La Nora, a small town in Murcia, Spain, to about 145 inhabitants [8]. Overall 101 questionnaires (from 47 men and 54 women) were included in the analyses. Electric field strength in the frequency range 1 MHz to 3 GHz was measured in the bedrooms of the participants. Data were analyzed in two different ways: first subjects were subdivided into those living less than 150 m from the base station and a second group living more than 250 m away (according to self-reports); the average exposure level of the first group was  $1.1 \text{ mW/m}^2$ , and of the second group  $0.1 \text{ mW/m}^2$ ; self-reported symptom severity was compared across these groups. The second method correlated log transformed field strengths with symptom scores. The majority of symptoms showed a relationship both by comparison of the contrast groups according to distance from the base station as well as when correlated to measured field strength. Strongest effects were observed for headaches, sleep disturbances, concentration difficulties, and discomfort.

In contrast to the French investigation the study has assessed actual exposure by short-term measurements in the bedrooms of participants. The fact that both, reported distance as well as measured field strength, correlated with symptom severity supports the hypothesis of an association between microwaves from the base station and wellbeing. However, because subjects knew that the intention of the study was to assess the impact of the base station there is a potential for bias. Also concerns of the participants about effects of the base station on health were not assessed. Furthermore, method of selection of participants was not reported.

In a cross-sectional study in the vicinity of 10 GSM base stations in rural and urban areas of Austria, Hutter et al. [9] selected 36 households randomly at each location based on the characteristics of the antennas. Selection was done in such a way as to guarantee a high exposure gradient. Base stations were selected out of more than 20 locations based on the following criteria: (1) at least 2 years operation of the antenna; (2) no protest against it before or after erection; (3) no nearby other base station; (4) transmission only in the 900 MHz frequency band. (The last two criteria were not fully met in the urban area.) In order to minimize intervention of interviewers all tests and questionnaires were presented on a laptop computer and subjects fulfilled all tasks on their own. Wellbeing was assessed by a symptoms list (v. Zerssen scale), sleeping problems by the Pittsburgh sleeping scale. In addition several tests of cognitive performance were applied. Concerns about environmental factors were inquired and sources of EMF exposure in the household were assessed as well. It was not disclosed to the subjects that the study was about the base station, but about environmental factors in general. Among other measurements high-frequency fields were assessed in the bedrooms. From the measured field strength of the BCCH maximum and minimum exposure to the base station signals were computed. In addition overall power density of all high-frequency fields was measured. Results of measurements from 336 households were available for analysis. Exposure from the base station was categorized into three ranges: below  $0.1 \text{ mW/m}^2$ , between  $0.1$  and  $0.5 \text{ mW/m}^2$ , and above  $0.5 \text{ mW/m}^2$ . Cognitive performance tended to be better at higher exposure levels and was statistically significant for perceptual speed after correction for confounders (age, gender, mobile phone use, and concerns about the base station). Subjective symptoms were generally more frequent at higher exposure levels and statistically increased prevalence was found for headaches, cold hands or feet, and concentration difficulties. Although participants reported more sleeping problems at higher exposure

levels, this effect was removed after controlling for concerns about the base station.

Despite limitations inherent in the cross-sectional study design the methodological problems mentioned in the French and Spanish investigations were avoided. Authors conclude: “The results of this study indicate that effects of very low but long lasting exposures to emissions from mobile telephone base stations on wellbeing and health cannot be ruled out. Whether the observed association with subjective symptoms after prolonged exposure leads to manifest illness remains to be studied.”

A study in employees working within or opposite a building with GSM base station antennas on the roof was reported by Abdel-Rassoul et al. [10]. The investigation took place in Shebin El-Kom City, Menoufiya Governorate, Egypt, where the first mobile phone base station was erected in 1998 on a building for agricultural professions. Overall 37 subjects working within this building and 48 subjects working in the agricultural directorate about 10 m opposite the building were considered exposed. A control group, working in another building of the agricultural administration located approximately 2 km away, consisted of 80 persons. Participants completed a structured questionnaire assessing educational and medical history. A neurological examination was performed and a neurobehavioral test battery (tests for visuomotor speed, problem solving, attention and memory) was presented. The combined exposed groups were compared to the control group that was matched by sex, age and other possible confounders. Statistical analysis accounted for these variables. Further comparisons were performed between subjects working in the building with the base station on the roof and those opposite. Exposed subjects performed significantly better in two tests of visuomotor speed and one test of attention, in two other tests the opposite was the case. The prevalence of headaches, memory problems, dizziness, tremors, depressive symptoms, and sleep disturbances was significantly higher among exposed inhabitants than controls. Measurements conducted 3 years before the investigation revealed compliance with the Egyptian standard ( $80 \text{ mW/m}^2$ ) with values between 27 and  $67 \text{ mW/m}^2$ , but locations of the measurements were not specified.

Like in the study of Hutter et al. [9] it was not disclosed to the participants that the study was about the base station. An important aspect is studying employees that occupy the area of exposure for 8–16 h a day. Several possible confounders (age, sex, education, smoking, and mobile phone use) were considered and did not change the reported results. Other factors like stressful working conditions, indoor pollutants and other attributes of the work place were not assessed and might have had an effect on the reported symptoms. Although no recent measurements were available it can be assumed that both, subjects working within the building as well as those opposite the building with the base station are exposed at comparatively high levels. The picture of one antenna shown in the article indicates that the panel is slightly uptilted. It can be assumed that the sidelobes of the antenna are directed

downwards into the building below the base station as well as into the opposite building. Measurements in Germany revealed that, in contrast to a general belief that there is no significant exposure in buildings below a base station antenna, the field strength in buildings below an antenna is almost equal to field strength in opposite buildings.

An experimental field trial was conducted in Bavaria [11] during three months before an UMTS antenna on a governmental building started operation. Based on a random sequence the antenna was turned on or off one, two, or three days in a row during 70 working days in winter 2003. Conditions were double-blind since neither the experimenters nor the participants knew whether the antenna was on or off. This was guaranteed by software manipulation of the antenna output that prohibited UMTS mobile phones from contacting the base station and by locating the computer controlling the antenna in a sealed room. The UMTS antenna operated at a mean frequency of 2167.1 MHz. The protocol has not been specified, but considering that no real transmission occurred it is assumed that only the service channel was used. The antenna had a down-tilt of  $8^\circ$  expected to result in rather high exposure within the building. Measured electric field strength in the rooms of the participants varied between the detection limit of the field probe ( $0.05 \text{ V/m}$ ) and  $0.53 \text{ V/m}$  (corresponding to  $0.75 \text{ mW/m}^2$ ) with an average of  $0.10 \pm 0.09 \text{ V/m}$  (corresponding to  $0.03 \text{ mW/m}^2$ ). Participants should answer an online questionnaire on each working day they were in the office in the morning when they arrived and in the evening shortly before leaving. The questionnaire consisted of a symptom list with 21 items, and in the evening participants should state whether or not they considered the antenna has been on during this day and whether they considered, if they experienced any adverse effects, these effects due to the base station. From approximately 300 employees working in the building 95 (28 females, 67 males) that answered the questionnaire on at least 25% of the working days were included in the analysis.

None of the 21 symptoms showed a statistically significant difference between days on and days off. A more comprehensive analysis of the overall score across all 21 items applying a mixed model with subjects as random factor and autoregressive residuals revealed a tendency ( $p = 0.08$ ) for an effect of actual exposure on the difference between morning and evening values. Self-rated electrosensitivity had a significant effect on evening scores but did not affect difference scores. As expected, subjective rating of exposure had a significant influence both, on evening scores and score difference. Correct detection rate of base station transmission mode was 50% and thus equal to chance. No person was able to detect operation mode correctly on significantly more days than expected.

The study design was a great strength of this pilot investigation. It combined the advantages of a field trial with the rigorous control of exposure conditions in an experiment. However, there are a number of severe shortcomings too: first, no correction for actual exposure has been applied. As

stated above, exposure varied considerably within the building and some participants were not exposed at detectable levels at all. The resulting exposure misclassification leads to a bias towards the null hypothesis. Furthermore, it was not specified which UMTS protocol was actually transmitted. Another important limitation is the quite low exposure even in the offices with the highest levels. Problems with the statistical evaluation are indicated by a highly significant time factor suggesting insufficient removal of autocorrelation. Finally, the symptom list contains several items that were not implicated previously as related to exposure from base stations (e.g. back pain). Such items reduce the overall power to detect an effect of base station exposure.

A cross-sectional study based on personal dosimetry was conducted in Bavaria [12]. In a sample of 329 adults (173 females, 155 males, and 1 unknown) chronic and acute symptoms were assessed. Chronic symptoms were taken from the Freiburger Beschwerdeliste and acute symptoms from the v. Zerssen list. Symptoms assessed were headache, neurological symptoms, cardiovascular symptoms, concentration problems, sleeping disorders and fatigue. Participants wore a dosimeter (Maschek ESM 140) for 24 h on the upper arm on the side used for holding a phone (during the night the dosimeter was placed next to the bed). The dosimeter measured exposure in frequency bands including GSM 900 up- and down-link, GSM 1800 up- and down-link, UMTS, DECT and WLAN (2.45 GHz).

Acute symptoms at noon and in the evening were dichotomized and related to exposure during the previous 6 h (night time measurements were considered biased and not analyzed). Exposure was expressed in percent of the ICNIRP reference levels. Odds ratios for the different symptom groups were computed in relation to exposure subdivided into quartiles with the first quartile as reference. Similarly, dichotomized chronic symptoms were related to average day time exposure levels. None of the symptom groups was significantly related to exposure. Odds ratios for headaches and cardiovascular symptoms during the last 6 months were increased for all three tested exposure quartiles (for headaches odds ratios were: 1.7, 2.7, and 1.2 for 2nd to 4th quartile; for cardiovascular symptoms these figures were 1.4, 3.3, and 2.4). But none of these odds ratios was statistically significant. Acute symptoms at noon and in the evening showed a tendency for lower prevalence of fatigue at higher exposure levels. Odds ratios for headaches and concentration problems in the evening were increased at higher exposure levels in the afternoon but also these results were statistically not significant (odds ratios for headaches were 1.7, 1.6, 3.1 and for concentration problems 1.4, 2.0, 1.4 for 2nd to 4th quartile of afternoon exposure levels).

Exposure was low and ranged from a daytime average of 0.05 V/m (at or below the limit of determination) to 0.3 V/m (corresponding to 0.24 mW/m<sup>2</sup> power density). (In order to make results comparable to other investigations figures expressed in percent of ICNIRP reference levels were recalculated to field strengths and power densities). Quartiles for

daytime exposure were: up to 0.075 V/m, 0.075 to 0.087 V/m, 0.087 to 0.110 V/m, and 0.110 to 0.3 V/m. It can be seen that the first three quartiles are almost indiscernible with a ratio of the upper limit of the third and first quartiles of only 1.5.

Although the study of Thomas et al. [12] was the first one using personal dosimetry in the context of investigating effects of exposure to mobile phone base station signals on wellbeing it has not explored the potential of an almost continuous exposure measurement. Only average exposure was computed and the probably most important nighttime values were left out. A number of different exposure metrics should have been assessed, like duration of exposure above a certain limit, maximum exposure level, longest period below limit of determination, and variability of exposure levels to name but a few. Furthermore, prevalence of symptoms was so low that the power of the investigation to detect even substantially increased risks was inferior (less than 25%). Despite these shortcomings the study has its merits as a first step in using personal dosimetry. An earlier report of the group [13] with a comparison between two personal dosimeters (Maschek and Antennessa) demonstrated that improvements are necessary before personal dosimetry can be successfully used in epidemiological studies.

A large population-based cross-sectional study was conducted in the context of the German 'Mobile Phone Research Program' in two phases [7]. In the initial phase 30,047 persons from a total of 51,444 (58% response rate) who took part in a nationwide survey also answered questions about mobile phone base stations. Additionally a list of 38 health complaints (Frick's list) was answered. Distance to the nearest base station was calculated based on geo-coded data of residences and base stations. In the second phase, all respondents (4150 persons) residing in eight preselected urban areas were contacted. In total, 3526 persons responded to a postal questionnaire (85% response rate) including questions about health concerns and attribution of symptoms to exposures from the base station as well as a number of standardized questionnaires: the Pittsburgh Sleep Quality Index, the Headache Impact Test, the v. Zerssen list of subjective symptoms, the profile of mental and physical health (SF 36), and a short version of the Trier Inventory of Chronic Stress. Overall 1808 (51%) of those that responded to the questionnaire agreed to have EMF measurement taken in their homes. Results of the large survey from the first phase of the study revealed a fraction of 10% of the population who attributed adverse health effects to the base station. An additional 19% were generally concerned about adverse effects of mobile phone base stations. Regression analysis of the symptoms summary score on distance to the base station (less or more than 500 m) and attribution/concerns about adverse effects adjusted for possible confounders (age, gender, SES, region and size of community) revealed a small but significant increase of the symptom score at closer distance to the base station. Higher effects, however, were obtained for concerns about adverse effects of the base station (with higher scores for those concerned) and still higher effects for



those that attributed their health problems to exposures from mobile phone base stations. The latter result is only to be expected because attribution presupposes existence of symptoms and hence those with attribution must have higher scores than those without. Because effects of concerns/attribution were accounted for in the multivariate model, effect of distance to the base station is independent of these concerns or attributions. In the second phase measurements in the bedrooms revealed an overall quite low exposure to EMFs from the base station. Only in 34% of the households was the exposure above the sensitivity limit of the dosimeters of  $0.05 \text{ V/m}$  ( $\sim 7 \mu\text{W/m}^2$ ). On average power density was  $31 \mu\text{W/m}^2$  and the 99th percentile amounted to  $307 \mu\text{W/m}^2$ . A dichotomization at the 90th percentile (exposure above  $0.1 \text{ V/m}$ , corresponding to  $26.5 \mu\text{W/m}^2$ ) did not indicate any effect of exposure on the different outcome variables but effects of attribution on sleep quality and overall symptom score (v. Zerssen list).

This large study has a number of important advantages: it started from a representative sample of the German population with over 30,000 participants and the second phase with a regional subsample had a participation rate of 85%. Furthermore, several well-selected standardized tests were used in the second phase. Results of the first phase are essentially in line with the Austrian study of Hutter et al. [9]. Not only the fraction with attribution of health complaints to exposure from the base station (10%) is identical, but also the higher symptom score in proximity to the base station independent of concerns/attributions found in the previous study has been replicated. However, the study has also severe shortcomings, most notably: the failure to include a sufficient number of participants that can be considered as exposed to microwaves from the base station. Note that Hutter et al. [9] selected households based on the characteristics of the antennas in such a way as to guarantee a large exposure gradient. In the randomly selected households of the study by Blettner et al. [7] the 90th percentile used as cutoff was well below the median ( $\sim 100 \mu\text{W/m}^2$ ) of the earlier investigation and the 99th percentile was still below the level ( $500 \mu\text{W/m}^2$ ) that was found to increase the prevalence of several symptoms. Therefore it is unlikely that the investigation of the second phase could detect an effect if it occurs at levels consistent with those reported by Hutter et al. [9].

## 2.2. Cancer

Despite considerable public concerns that exposure to microwaves from mobile phone base stations could be detrimental to health and may, in particular, cause cancer, up to now only two studies of cancer in the vicinity of base stations applying basically an ecological design have been published.

In a Bavarian town, Neila, the physicians of the town conducted an epidemiological investigation [14] to assess a possible association between exposure to base station radiation and cancer incidence. The design used was an improved ecological one. Two study areas were defined: one within

a circle of 400 m radius around the only base stations (two that were located in close proximity to each other) of the town, and one area further than 400 m from the base stations. Within these defined areas streets were randomly selected (after exclusion of a street where a home for retired people was situated) and all general practitioners of the town that were active during the whole period of operation of the base stations (one base station started operation September 1993 the other December 1997) scanned their files for patients living in the selected streets. Overall 967 individuals were found, constituting approximately 90% of the reference population. The study period 1/1994 to 3/2004 was subdivided into two segments: The first 5 years of operation of the base station (1994 through 1998) and the period from the sixth year, 1999, until 3/2004. Among the identified individuals 34 incident cases of cancer (excluding non-melanoma skin cancer) were found. Assessment of cancer cases was assumed to be complete and all cases were verified histologically and by hospital discharge letters (note that there is no cancer registry in Bavaria). Age distribution was similar in the two areas with a mean age of 40.2 years in both, the area within 400 m of the base station and the area further apart. Crude annual cancer incidence in the first 5 years after start of operation of the base station was  $31.3 \times 10^{-4}$  and  $24.7 \times 10^{-4}$  in the closer and farther area, respectively. In the second period these figures were  $76.7 \times 10^{-4}$  and  $24.7 \times 10^{-4}$ . The age and gender adjusted expected value of incident cancer cases in the study population based on data from Saarland, a German county with a cancer registry, is  $49 \times 10^{-4}$ . In the second period cancer incidence in the area within 400 m of the base station was significantly elevated, both, compared to the area further away as well as compared to the expected background incidence. The incidence in the region further apart was reduced but not significantly when compared to the expected value.

Although this so-called Neila-study applied an improved ecological design with a random selection of streets and inclusion of some information from selected individuals, it is still subject to potential bias because relevant individual risk factors could not be included in the analyses.

A similar though less rigorous study has been performed in Netanya, Israel. Wolf and Wolf [15] selected an area 350 m around a base station that came into operation 7/1996. The population within this area belongs to the outpatient clinic of one of the authors. The cohort within this area consisted of 622 people living in this area for at least 3 years at study onset, which was one year after start of operation of the base station and lasted for 1 year. Overall cancer incidence within the study area was compared to a nearby region, to the whole city of Netanya, and to national rates. In the second year after onset of operation 8 cancer cases were diagnosed in the study area. In the nearby area with a cohort size of 1222 individuals, 2 cases were observed. Comparison to the total population with an expected incidence of  $31 \times 10^{-4}$  indicates a pronounced increase in the study area with an incidence of  $129 \times 10^{-4}$ . Also against the whole town of Netanya an increased incidence was noted especially in women. In an

addendum authors noted that also in the subsequent year 8 new cases were detected in the study area while in the period 5 years before the erection of the base station 2 cases occurred annually. Spot measurements of high frequency fields were conducted in the homes of cancer cases and values between 3 and 5 mW/m<sup>2</sup> were obtained. Although these values are well below guideline levels, they are quite high compared to typical values measured in randomly selected homes [7].

Also in the case of the Netanya study lack of information on individual risk factors makes interpretation difficult. Furthermore, migration bias has not been assessed although only subjects were included that occupied the area for at least 3 years. The short latency after start of operation of the base station rules out an influence of exposure on induction period of the diseases. The substantial increase of incidence is also hardly explainable by a promotional effect.

### 3. Experimental studies

#### 3.1. Experiments in human sensitive and non-sensitive individuals

There are persons who claim to suffer from immediate acute as well as chronic effects on exposure to EMF and in particular to those from mobile phones or their base stations. Often these persons are called EMF hypersensitive (EHS). The preferred term agreed upon at a WHO workshop [16] was Idiopathic Environmental Intolerance with attribution to EMF (IEI-EMF). Indeed, it would be a misunderstanding to confuse EHS with allergic reactions; rather these persons react with different unspecific symptoms such as headaches, dizziness, loss of energy, etc. Whether these persons have actually the ability to tell the difference between situations with and without exposure to EMFs is an open question. In a recent review Rösli [17] concluded that "...the large majority of individuals who claim to be able to detect low level RF-EMF are not able to do so under double-blind conditions. If such individuals exist, they represent a small minority and have not been identified yet." However, it is important to differentiate between EMF sensitivity and sensibility [18]. Independent of the question whether or not there are individuals that sense the presence of low levels of EMFs such as those measured in homes near mobile phone base stations, there could well be an effect of such exposures on wellbeing and performance even under short-term exposure conditions. In several experimental investigations this question has been addressed by exposure of persons with self-reported symptoms and also in persons without known adverse reaction to an assumed exposure.

The first of these investigations was carried out by the Netherlands Organization for Applied Scientific Research (TNO) and published as a research report [19]. Two groups of persons were included in the experiment. One group consisted of individuals (25 females, 11 males) who have previously reported complaints and attributed them to GSM

exposure. The other group consisted of subjects without such complaints (14 females, 22 males). Four experimental conditions were applied in a double-blind fashion: Sham exposure, exposure to 945 MHz GSM, 1840 MHz GSM, and 2140 MHz UMTS. Each participant underwent sham exposure and two of the active exposure conditions. Sequence of exposure was balanced such that each active exposure condition was tested equally often at each of three experimental sessions. Each experimental session and a training session lasted for 45 min. All three experimental sessions and the training session were completed on one day for each participant. Both, for GSM and UMTS exposure, a base station antenna was used and a simulated base station signal was transmitted during sessions. For the GSM conditions a 50% duty cycle (4 slots occupied) was applied with pulses of peak amplitudes of 1 V/m (0.71 V/m effective field strength; corresponding to 1.3 mW/m<sup>2</sup>). For UMTS exposure a protocol was used with different low frequency components and an effective field strength of 1 V/m (corresponding to 2.7 mW/m<sup>2</sup>). During each session several performance tests were conducted and immediately after each session a wellbeing questionnaire was administered (an adapted version of the Quality-of-Life Questionnaire of Bulpitt and Fletcher [20] with 23 items).

Overall score of wellbeing was significantly reduced in both groups after the UMTS condition compared to sham exposure. Considering subscores anxiety symptoms, somatic symptoms, inadequacy symptoms, and hostility symptoms were increased in the groups of sensitive individuals whereas in the control group only inadequacy symptoms were increased after UMTS exposure compared to sham. No effects were found in the two GSM exposure conditions. Concerning cognitive performance both groups revealed significant exposure effects in almost all tests in different exposure conditions. In most of these tests reaction time was reduced except for one simple reaction time task.

This study had an enormous echo both in the media as well as in the scientific community because it was the first experimental investigation with very low exposure to base station like signals and in particular to UMTS signals, and because it was conducted by a highly respected research institution reporting systematic effects of exposure that seemed to support citizens initiatives claiming that base stations have adverse effects on wellbeing and health. Immediately doubts were expressed that results could be biased due to a faulty methodology. In fact, study design can be improved. First of all testing all exposure conditions on the same day has the advantage to reduce variance from between day differences but could cause transfer effects if biological reactions do not immediately terminate after end of exposure and start of the next condition. Also time-of-day effect from chronobiological variations could be superimposing the reactions from exposure. Such effects are sometimes not removed by balancing exposure conditions. Second, not all subjects were tested under all exposure conditions. The decision to reduce total experimental duration by presenting only two of the three exposure conditions together with sham was sound but



on the other hand led to a reduced power. Several other arguments such as the different gender distribution in the two groups are not very important because each subject served as his/her own control and comparison between groups was not important in this investigation. Other criticism was expressed against statistical analysis. No correction for multiple testing was applied. While some advice protection against inflation of type I error others recommend correction only for crucial experiments and not for pilot studies like this. Another, more serious, criticism was put forward against disregarding sequence of experimental conditions. As mentioned above, sequence, transfer, and time-of-day effects could have compromised results because such effects are not completely removed by balancing exposure sequence. Due to this criticism several studies were planned that should investigate whether the effects observed in the TNO study are robust and could be replicated under improved study designs.

One of these experiments was performed in Switzerland [21]. Like in the TNO study, two groups of individuals were included: one with self-reported sensitivity to RF-EMF (radio-frequency EMF) and a reference group without complaints. The first group consisted of 33 persons (19 females, 14 males) and the reference group of 84 persons (43 females, 41 males). The experiment consisted of three experimental and one training session each 1 week apart performed on the same time of day ( $\pm 2$  h). Design was a randomized double-blind cross-over design like in the case of the TNO study, however, with a week between sessions and with all subjects tested under all experimental conditions that were solely simulated UMTS base station exposure at 1 V/m, 10 V/m and sham. The same UMTS protocol as in the TNO study was used. Each exposure condition lasted for 45 min. During exposure two series of cognitive tasks were performed. After each exposure condition the same questionnaire as has been used in the TNO study was applied and questions about sleep in the previous night, alcohol, coffee consumption, etc., were asked. Moreover, subjects had to rate the perceived field strength of the previous exposure condition on a visual analogue scale. In addition, before and after each session the short Questionnaire on Current Disposition [22] was answered by participants. Questionnaires were presented in a separate office room.

Except for a significant reduction of performance speed of sensitive participants in the 1 V/m condition in one of six cognitive tests no effect of exposure was detected. In particular, no reduction of wellbeing neither as assessed by the TNO questionnaire nor from scores of the Questionnaire on Current Disposition was found. Also correlation between perceived and real exposure was not more often positive than expected from chance. Fig. 2 compares results of the TNO study and the results of Regel et al. [21] for the matching conditions (UMTS at 1 V/m). There are some notable differences between the two studies: first, the reference group in the study of Regel et al. [21] had significantly higher scores (reduced wellbeing) as the reference group in the TNO study in both the sham and the UMTS 1 V/m condition; second,

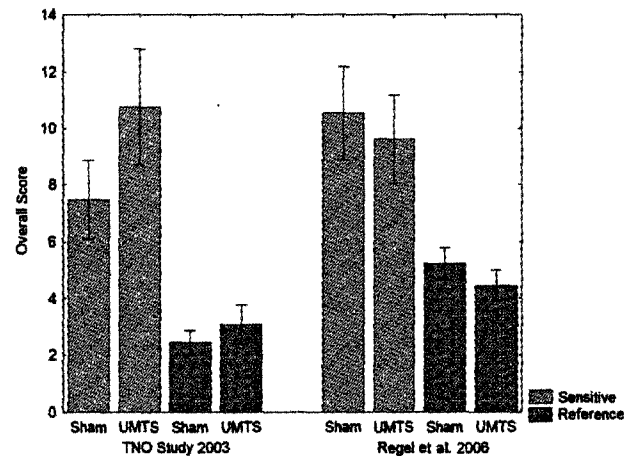


Fig. 2. Comparison of mean ( $\pm$ SEM) overall wellbeing scores (TNO questionnaire) obtained in the TNO study [19] and in the study of Regel et al. [21] for the matching conditions: Sham exposure and UMTS exposure at 1 V/m in sensitive participants and the reference group.

average scores from sensitive participants after exposure at 1 V/m are comparable in both studies but the sham condition resulted in much lower scores (better wellbeing) in the TNO study. There are several explanations for this difference between the two studies. It is possible that the reference group in the TNO study consisted of exceptionally robust individuals. The fraction of males was higher in the TNO study and males have typically lower scores. However, considering that the reference group in the TNO study was almost 10 years older (mean age 47 years) as compared to the study of Regel et al. [21] (mean age 38 years) this is not a satisfactory explanation. It is possible that the basic adversity of the experimental setup was higher in the latter study resulting in overall greater reduction of wellbeing. That this has not been observed in the sensitive group assumed to be more vulnerable to a 'nocebo' effect (the nocebo effect is the inverse of the placebo effect describing a situation when symptoms occur due to expecting adverse reactions) in both conditions could be due to a ceiling phenomenon. Although the study by Regel et al. [21] had an improved design and could not replicate the earlier findings of the TNO study, doubts exist whether this can be considered a refutation of an effect of UMTS exposure on wellbeing.

Another experimental study in sensitive and non-sensitive participants has been conducted in Essex, Great Britain, by Eltiti et al. [23]. The experiment consisted of two phases: an open provocation test and a series of double-blind tests. In the open provocation phase 56 self-reported sensitive and 120 non-sensitive control individuals participated. Of these, 44 sensitive (19 females, 25 males) and 115 controls (49 females, 66 males) also completed the double-blind tests. Participants took part in four separate sessions each at least 1 week apart. First session was the open provocation trial, sessions 2–4 were double-blind exposure trials with a sham, a GSM and a UMTS exposure condition. Double-blind sessions were reported to last for 1.5 h, however, Table 1 of the

article showed an overall length of 48 min only. GSM exposure was a simulated base station signal with both a 900 and a 1800 MHz component each at an average level of 5 mW/m<sup>2</sup> and with a simulated BCCH with all time slots occupied and a TCH with a simulated 40% call activity resulting in a total of 10 mW/m<sup>2</sup> GSM exposure at the position of the participants (corresponding to 1.9 V/m E-field strength). The UMTS signal had a frequency of 2020 MHz with a power flux density of 10 mW/m<sup>2</sup> over the area where the participant was seated. Traffic modeling for the UMTS signal was achieved using a test model representing a realistic traffic scenario, with high peak to average power changes. During double-blind sessions participants watched a BBC “Blue Planet” video for 20 min, performed a mental arithmetic task for 20 min, performed a series of cognitive tasks lasting 8 min, and made ‘on/off’ judgments. During the first 40 min every 5 min subjective wellbeing was recorded on visual analogue scales (VAS) measuring anxiety, tension, arousal, relaxation, discomfort, and fatigue. In addition a symptom scale consisting of 57 items was answered. During the whole period physiological measurements of heart rate, blood volume pulse, and skin conductance were performed.

Physiological measurements revealed higher average values for sensitive individuals compared to controls which were especially high under UMTS exposure conditions. Symptom list did not reveal any differences between double-blind conditions, but the overall frequency of solicited symptoms was low. Concerning subjective wellbeing as assessed by VAS there were increased values for anxiety, tension, and arousal under GSM and especially UMTS exposure conditions. Combining all scores of the six scales (with relaxation reflected) reveals a significant increase during UMTS exposure compared to sham for the sensitive group and a significant reduction for the control group (see Fig. 3). Judgment of participants about presence of exposure was not correct more often than inferred from chance.

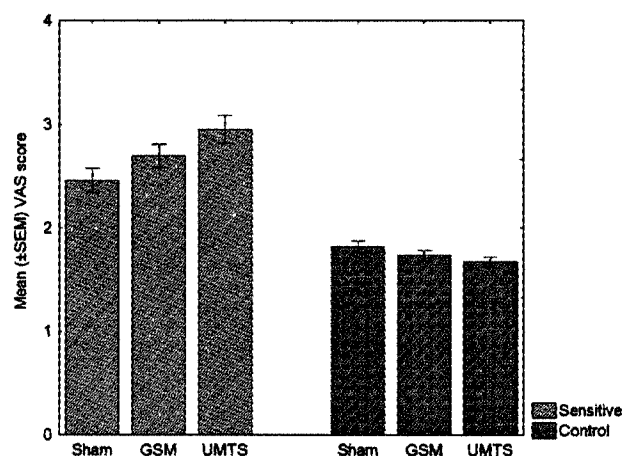


Fig. 3. Mean ( $\pm$ SEM) total visual analogue scale scores computed from Table 2 of Eltiti et al. [23] during sham, GSM, or UMTS exposure in sensitive and control individuals.

The increased values for anxiety, tension, and arousal found in this investigation were interpreted by the authors as due to an imbalance in the sequence of conditions with UMTS being more often the first exposure condition presented in the double-blind sessions. The imbalance was due to not reaching the predefined sample size. This points to the importance of setting the block size for randomization to a low level (e.g. in this experiment with 6 possible exposure sequences a block size of 18 would have been appropriate). Interpretation of authors, however, is questionable as pointed out by Rösli and Huss [24]. For arousal tabulated values stratified for sequence of presentation (Table 3 in [23]) demonstrates that the difference between sham and UMTS is present regardless of sequence of presentation. An additional analysis of the authors presented in response to the criticism in their statistical analysis seems to support their view that the observed difference to sham is due to a sequence effect. However, it seems that this analysis has not been correctly applied as the sequence was introduced as a between subjects factor which corrects only the interaction between group and condition. Also the figure they provided [23] is inconclusive as it only demonstrates what is already known: that first exposure leads to higher reduction of wellbeing (higher values of arousal). This investigation, although well designed and applying a more realistic exposure scenario than the other two studies, leaves some questions open. Despite an apparent corroboration of the findings of the TNO study, the imbalance in the sequence of exposures makes it difficult to decide whether the interpretation of authors that the observed effect is due to an excess number of UMTS exposures presented first in the sequence is correct or an actual effect occurred. Irrespective of these difficulties, consistent with the other investigations, wellbeing was not strongly affected.

There are several other investigations of a similar type that have been completed and already reported at scientific meetings (e.g. Watanabe, Japan; Augner, Austria, personal communication) but have not yet been published.

### 3.2. Animal and in vitro experiments

Anane et al. [25] applied the DMBA (7,12-dimethylbenz(a)anthracene) model of mammary tumor induction in female Sprague–Dawley rats to test whether a sub-chronic exposure to microwaves from a GSM-900 base station antenna affects tumor promotion or progression. Exposure was 2 h/day, 5 days/week for 9 weeks starting 10 days after application of 10 mg DMBA administered at an age of animals of 55 days. Exposure was applied in an anechoic chamber with animals placed in Plexiglas compartments that confined animals to a position parallel to the E-field. Details of the exposure protocol were not provided. Two series of experiments were conducted with four groups of 16 animals each. In the first experiment groups were: sham, 1.4, 2.2, and 3.5 W/kg whole-body SAR, and the second experiment with sham, 0.1, 0.7, and 1.4 W/kg. In the first experiment the tumor incidence rate was significantly increased at 1.4

and 2.2 W/kg exposure, while in the second experiment the incidence at 1.4 W/kg was significantly reduced.

The experiment by Anane et al. [25] is inconclusive not only because of the divergent results of the two experiments at the same exposure condition (1.4 W/kg SAR) but mainly because of the insufficient size of experimental groups. With a 70% background tumor incidence as observed in this investigation even for an increase to 100% in the exposed group the power to detect this difference at a significance level of 5% is less than 60%. Furthermore, considering experimental and biological variation substantial differences may occur by chance simply due to different distribution of background risk between experimental groups. Therefore, in contrast to the statement of authors that relevant differences would be detected with 16 animals per group, the study was severely underpowered and prone to spurious effects from uneven distribution of background risk. Also stress from confinement of animals could have contributed to the ambiguous results.

Yurekli et al. [26] report an experiment in male Wistar albino rats with the aim to analyze oxidative stress from whole-body exposure to a GSM 945 MHz signal at a SAR level of 11.3 mW/kg. In a gigahertz transverse (GTEM) cell a base station exposure in the far field was simulated. Two groups of rats, 9 animals in each group, were either exposed 7 h a day for 8 days or sham exposed. At the end of the exposure blood was withdrawn and malondialdehyde (MDA), reduced glutathione (GSH), and superoxide dismutase (SOD) were measured. MDA as well as SOD was significantly increased after exposure compared to sham, while GSH was significantly reduced. These results indicate that exposure may enhance lipid peroxidation and reduce the concentration of GSH which would increase oxidative stress. A disadvantage in this experiment was that the experiments were carried out sequentially and therefore animals differed in weight and no blinding could be applied.

In a series of experiments conducted in the Kashima Laboratory, Kamisu, Japan, different *in vitro* assays were applied to test whether irradiation with 2.1425 GHz, which corresponds to the middle frequency allocated to the down-link signal of IMT-2000 (International Mobile Telecommunication 2000, a 3G wide-band CDMA system), leads to cellular responses relevant for human health [27–29]. In the first experiment phosphorylation and gene expression of p53 was assessed [27]. In the second experiment heat-shock protein expression was evaluated in the human glioblastoma cell line A172 and human IMR-90 fibroblasts [28]. The effect of exposure of BALB/T3T cells on malignant transformation, on promotion in MCA (3-methylcholanthrene) treated cells, and on co-promotion in cells pretreated with MCA and co-exposed to TPA (12-O-tetradecanoylphorbol-13-acetate) was investigated by Hirose et al. [29]. In none of these experiments applying the same exposure regimen but different intensities and exposure durations (80 mW/kg SAR up to 800 mW/kg SAR, 2 h to several weeks) an effect of exposure was observed. Exposure facility comprised of two anechoic chambers allowing blinded simultaneous exposure of an array

of  $7 \times 7$  dishes in each chamber. Dishes were placed in a culture cabinet located in the anechoic chamber and exposed to radiation from a horn antenna whose signals were focused by a dielectric lens to obtain homogenous irradiation of the dishes. Details of the exposure protocol were not disclosed. It is stated that an IMT-2000 signal at a chiprate (a chip is a byte of information) of 3.84 Mcps was used for exposure. Assuming that it did not contain any low-frequency components as typically present in actual exposures the implications of the findings are unclear. It is rarely supposed that the high-frequency components of RF-EMFs itself are able to elicit any relevant effects in the 'low-dose' range. Rather low-frequency modulation may contribute to biological responses. Therefore, results of these Japanese investigations are of limited value for risk assessment, conditional on them having no such biologically relevant exposure attributes.

#### 4. Discussion

Although there is considerable public concern about adverse health effects from long-term exposure to microwaves from mobile phone base stations there are only few studies addressing this issue. Several reasons can be identified for the scarcity of scientific investigations. First of all, WHO has discouraged studies of base stations, at least concerning cancer as endpoint, because retrospective assessment of exposure was considered difficult. Also COST 281 did not recommend studies of base stations and stated in 2002: "If there is a health risk from mobile telecommunication systems it should first be seen in epidemiological studies of handset use."

It is not appreciated that there are substantial and important differences between exposure to handsets and base stations. The typically very low exposure to microwaves from base stations, rarely exceeding  $1 \text{ mW/m}^2$ , was deemed very unlikely to produce any adverse effect. Assuming energy equivalence of effects a 24 h exposure at  $1 \text{ mW/m}^2$  from a base station would be roughly equivalent to 30 min exposure to a mobile phone operating at a power of 20 mW (average output power in areas of good coverage). Because we do not know whether time-dose reciprocity holds for RF-EMF and whether there is a threshold for biological effects, there is no *a priori* argument why such low exposures as measured in homes near base stations could not be of significance for wellbeing and health. As an example from a different field of environmental health consider noise exposure: it is well known that at noise levels exceeding 85 dB(A) a temporary shift of hearing threshold occurs and that, besides this short-term effect, after years of exposure noise induced hearing loss may occur. On the other hand, at a sound pressure of more than a factor of 1000 below, when exposure occurs during the night, exposed individuals will experience sleep disturbances that could affect health in the long run. From this example it follows that exposure may have qualitatively different effects at different exposure levels.

The most important difference between mobile phone use and exposure from base station signals is duration of exposure. While mobile phones are used intermittently with exposure duration seldom exceeding 1 h per day, exposure to base stations is continuous and for up to 24 h a day. It has also to be mentioned that the exposure of mobile phone users is in the near field and localized at the head region, while base stations expose the whole body to the far field. Strictly speaking exposure from mobile phones and their base stations have almost nothing in common except for the almost equal carrier frequency that is likely of no importance for biological effects.

Concerning reconstruction of exposure to base station signals there is no greater difficulty than for retrospective assessment of exposure to mobile phones. It is not always necessary to determine exposure precisely. For epidemiological investigations it often suffices to have a certain gradient of exposures. As long as any two persons can be differentiated along such a gradient epidemiological investigations can and should be carried out.

There are seven field studies of wellbeing and exposure to base station signals available to date. Two were in occupational groups working in a building below [11] or below as well as opposite a building with a roof-mounted base station antenna [10]. The other five were in neighbors of base stations: Santini et al. [5,6], Navarro et al. [8], Hutter et al. [9], Blettner et al. [7], and Thomas et al. [12]. Studies had different methodologies with the least potential for bias in the studies of Hutter et al. [9] and Blettner et al. [7]. All other studies could be biased due to self-selection of study participants. One study explored personal dosimetry during 24 h [12] but results were inconclusive due to insufficient power and omission of nighttime measurements. The study of Blettner et al. [7] had an interesting design with a first phase in a large population based representative sample and a second phase with individual measurements in the bedrooms of participants that were a subgroup of the larger sample. Unfortunately this second sample did not contain a sufficiently large fraction of individuals with relevant exposure (99% had bedside measurements below  $0.3 \text{ mW/m}^2$ ).

Despite some methodological limitations of the different studies there are still strong indications that long-term exposure near base stations affects wellbeing. Symptoms most often associated with exposure were headaches, concentration difficulties, restlessness, and tremor. Sleeping problems were also related to distance from base station or power density, but it is possible that these results are confounded by concerns about adverse effects of the base station, or more generally, by specific personality traits. While the data are insufficient to delineate a threshold for adverse effects the lack of observed effects at fractions of a  $\text{mW/m}^2$  power density suggests that, at least with respect to wellbeing, around  $0.5\text{--}1 \text{ mW/m}^2$  must be exceeded in order to observe an effect. This figure is also compatible with experimental studies of wellbeing that found effects at  $2.7$  and  $10 \text{ mW/m}^2$ .

There are regular media reports of an unusually high incidence of cancer in the vicinity of mobile phone base stations. Because there are several hundred thousand base stations operating all over the world some must coincide by chance with a high local cancer incidence. Regionally cancer incidence has a distribution with an overdispersion compared to the Poisson distribution. Overdispersion is predominantly due to variations in the distribution of age and gender. Therefore, a much higher number of cases than expected from average incidences can occur by chance. Unfortunately there are no multi-regional systematic investigations of cancer incidence related to mobile phone base stations available to date. Only studies in a single community, one in Bavaria [14] and one in Israel [15], have been published that reported a significantly increased incidence in an area of  $400$  and  $350 \text{ m}$  around a base station, respectively. Although incidence in proximity to the base station strongly exceeded the expected values and was significant even considering overdispersion in the case of the Neila study in Bavaria, still no far reaching conclusions can be drawn due to the ecological nature of the studies. However, both studies underline the urgent need to investigate this problem with an appropriate design. Neubauer et al. [30] have recommended focusing initially on short-term effects and 'soft' outcomes given the problems of exposure assessment. However, as has been mentioned previously, the problems of exposure assessment are less profound as often assumed. A similar approach as chosen in the study of leukemia around nuclear power plants [31] could be applied also for studying cancer in relation to base station exposure. Such a case-control design within areas around a sufficiently large sample of base stations would provide answers to the questions raised by the studies of Eger et al. [14] and Wolf and Wolf [15].

In 2003 the so-called TNO study [19] had received wide publicity because it was the first experimental investigation of short-term base station exposure in individuals that rated themselves sensitive to such signals. A lot of unfounded criticism was immediately raised such as complaints about the limited sample size and the not completely balanced design. But also valid arguments have been put forward. The consecutive tests with all experimental conditions presented one after the other could result in sequential effects that may not be completely removed by balancing the sequence of exposures. In several countries follow-up studies were initiated two of which have already been published [21,23]. One of these experiments partly supported the TNO study the other found no effect. While the study of Regel et al. [21] closely followed the conditions of the previous experiment only avoiding the shortcomings of a sequential within-day design and improvements by including two intensities of UMTS exposure, the study of Eltiti et al. [23] had a different procedure and included physiological measurements. Regel et al. [21] applied the same questionnaire as has been used in the TNO study. Because non-sensitive participants and sensitive participants during sham exposure (despite their almost 10 years younger age) reported considerably lower wellbeing,

it is possible that the experimental setup was more adverse and imposed too much stress such that these conditions confounded the effect of the base station exposure. Results of the other replication experiment of Eltiti et al. [23] may be compromised by an imbalance in the sequence of experiments with more sensitive participants receiving UMTS exposure in the first session. Hence, based on available evidence, it cannot be firmly decided whether such weak signals as applied in these experiments to simulate short-term base station exposure affects wellbeing.

Concerning animal experiments and in vitro investigations the data base is insufficient to date. While in vivo exposure of Wistar albino rats [26] imply an induction of oxidative stress or an interaction with antioxidant cellular activity, in vitro experiments [27] found no indication of cellular stress in human glioblastoma cells and fibroblasts. While some may be inclined to attribute effects in the low-dose range to experimental errors there is the possibility that the characteristics of the exposure that are relevant for an effect to occur simply vary in the experiments and lead to ambiguous results. As long as these decisive features of the exposure (if they actually exist) are unknown and in particular the type and components of low-frequency modulation vary across experiments, it is impossible to coherently evaluate the evidence and to come to a science based conclusion.

Overall results of investigations into the effects of exposure to base station signals are mirroring the broader spectrum of studies on handsets and on RF-EMF in general. There are indications from epidemiology that such exposures affect wellbeing and health weakly supported by human provocation studies and an inconclusive body of evidence from animal and in vitro studies.

## References

- [1] N. Leitgeb, J. Schröttner, M. Böhm, Does “electromagnetic pollution” cause illness? An inquiry among Austrian general practitioners, *Wien. Med. Wochenschr.* 155 (9–10) (2005) 237–241.
- [2] A. Huss, M. Röösli, Consultations in primary care for symptoms attributed to electromagnetic fields—a survey among general practitioners, *BMC Public Health* 30 (6) (2006) 267.
- [3] M. Kundi, F. Schinner, W. Mosgöller, H.-P. Hutter, Proceedings of the International Workshop on Possible Biological and Health Effects of RF Electromagnetic Fields, 25–28 October, University of Vienna, 1998.
- [4] WHO 2006, WHO Research Agenda for Radio Frequency Fields.
- [5] R. Santini, P. Santini, J.M. Danze, P. Le Ruz, M. Seigne, Enquête sur la santé de riverains de stations relais de téléphonie mobile: I/Incidences de la distance et du sexe, *Pathol. Biol. (Paris)* 50 (2002) 369–373.
- [6] R. Santini, P. Santini, J.M. Danze, P. Le Ruz, M. Seigne, Enquête sur la santé de riverains de stations relais de téléphonie mobile: II/Incidences de l’âge des sujets, de la durée de leur exposition et de leur position par rapport aux antennes et autres sources électromagnétiques, *Pathol. Biol. (Paris)* 51 (2003) 412–415.
- [7] M. Blettner, B. Schlehofer, J. Breckenkamp, B. Kowall, S. Schmiedel, U. Reis, P. Potthoff, J. Schüz, G. Berg-Beckhoff, Querschnittstudie zur Erfassung und Bewertung möglicher gesundheitlicher Beeinträchtigungen durch die Felder von Mobilfunkbasisstationen, *BfS* (2007).
- [8] E.A. Navarro, J. Segura, M. Portoles, C. Gomez-Perretta de Mateo, The microwave syndrome: a preliminary study in Spain, *Electromag. Biol. Med.* 22 (2003) 161–169.
- [9] H.-P. Hutter, H. Moshammam, P. Wallner, M. Kundi, Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations, *Occup. Environ. Med.* 63 (2006) 307–313.
- [10] G. Abdel-Rassoul, O. Abou El-Fateh, M. Abou Salem, A. Michael, F. Farahat, M. El-Batanouny, E. Salem, Neurobehavioral effects among inhabitants around mobile phone base stations, *Neurotoxicology* 28 (2) (2006) 434–440.
- [11] S. Heinrich, A. Ossig, S. Schlittmeier, J. Hellbrück, Elektromagnetische Felder einer UMTS-Mobilfunkbasisstation und mögliche Auswirkungen auf die Befindlichkeit—eine experimentelle Felduntersuchung, *Umwelt. Med. Forsch. Prax.* 12 (2007) 171–180.
- [12] S. Thomas, A. Kühnlein, S. Heinrich, G. Praml, D. Nowak, R. von Kries, K. Radon, Personal exposure to mobile phone frequencies and well-being in adults: a cross-sectional study based on dosimetry, *Bioelectromagnetics* 29 (2008) 463–470.
- [13] K. Radon, H. Spiegel, N. Meyer, J. Klein, J. Brix, A. Wiedenhofer, H. Eder, G. Praml, A. Schulze, V. Ehrenstein, R. von Kries, D. Nowak, Personal dosimetry of exposure to mobile telephone base stations? An epidemiologic feasibility study comparing the Mashek dosimeter prototype and the Antennessa SP-090 system, *Bioelectromagnetics* 27 (2006) 77–81.
- [14] H. Eger, K.U. Hagen, B. Lucas, P. Vogel, H. Voit, Einfluss der räumlichen Nähe von Mobilfunkseendeanlagen auf die Krebsinzidenz, *Umwelt-Medizin-Gesellschaft* 17 (2004) 273–356.
- [15] R. Wolf, D. Wolf, Increased incidence of cancer near a cellphone transmitter station, *Int. J. Cancer Prev.* 1 (2004) 123–128.
- [16] K. Hansson Mild, M. Repacholi, E. van Deventer, P. Ravazzani (Eds.), 2006. Working Group Report. In: *Proceedings International Workshop on EMF hypersensitivity*, 25–27 October 2004, Prague, Czech Republic. Milan: WHO Press, pp. 15–26. Available from: [http://www.who.int/peh-emf/publications/reports/EHS\\_Proceedings\\_June2006.pdf](http://www.who.int/peh-emf/publications/reports/EHS_Proceedings_June2006.pdf), accessed August 2008.
- [17] M. Röösli, Radiofrequency electromagnetic field exposure and non-specific symptoms of ill health: a systematic review, *Environ. Res.* 107 (2008) 277–287.
- [18] N. Leitgeb, J. Schröttner, Electrosensitivity and electromagnetic hypersensitivity, *Bioelectromagnetics* 24 (2003) 387–394.
- [19] A.P.M. Zwamborn, S.H.J.A. Vossen, B.J.A.M. van Leersum, M.A. Ouwers, W.N. Mäkel, Effects of Global Communication System Radio-Frequency Fields on Well being and Cognitive Functions of Human Subjects with and without Subjective Complaints. FEL-03-C148, TNO, The Hague, the Netherlands, 2003.
- [20] C.J. Bulpitt, A.E. Fletcher, The measurement of quality of life in hypertensive patients: a practical approach, *Br. J. Clin. Pharmacol.* 30 (1990) 353–364.
- [21] S.J. Regel, S. Negovetic, M. Röösli, V. Berdinas, J. Schuderer, A. Huss, U. Lott, N. Kuster, P. Achermann, UMTS base station like exposure, well being and cognitive performance, *Environ. Health Perspect.* 114 (2006) 1270–1275.
- [22] B. Müller, H.D. Basler, Kurzfragebogen zur Aktuellen Beanspruchung (KAB), Beltz, Weinheim, Germany, 1993.
- [23] S. Eltiti, D. Wallace, A. Ridgewell, K. Zougkou, R. Russo, F. Sepulveda, D. Mirshekar-Syahkal, P. Rasor, R. Deeble, E. Fox, Does short-term exposure to mobile phone base station signals increase symptoms in individuals who report sensitivity to electromagnetic fields? A double-blind randomised provocation study, *Environ. Health Perspect.* 115 (2007) 1603–1608.
- [24] M. Röösli, A. Huss, Mobile phone base station exposure and symptoms, *Environ. Health Perspect.* 116 (2008) A62–A63.
- [25] R. Anane, P.E. Dulou, M. Taxile, M. Geffard, F.L. Crespeau, B. Veyret, Effects of GSM-900 microwaves on DMBA-induced mammary gland tumors in female Sprague–Dawley rats, *Radiat. Res.* 160 (2003) 492–497.

- [26] A.I. Yurekli, M. Ozkan, T. Kalkan, H. Saybasili, H. Tuncel, P. Atukeren, K. Pinar, K. Gumustas, S. Seker, GSM base station electromagnetic radiation and oxidative stress in rats, *Electromag. Biol. Med.* 25 (2006) 177–188.
- [27] H. Hirose, N. Sakuma, N. Kaji, T. Suhara, M. Sekijima, T. Nojima, J. Miyakoshi, Phosphorylation and gene expression of p53 are not affected in human cells exposed to 2.1425 GHz band CW or W-CDMA modulated radiation allocated to mobile radio base stations, *Bioelectromagnetics* 27 (2006) 494–504.
- [28] H. Hirose, N. Sakuma, N. Kaji, K. Nakayama, K. Inoue, M. Sekijima, T. Nojima, J. Miyakoshi, Mobile phone base station emitted radiation does not induce phosphorylation of hsp27, *Bioelectromagnetics* 28 (2007) 99–108.
- [29] H. Hirose, T. Suhara, N. Kaji, N. Sakuma, M. Sekijima, T. Nojima, J. Miyakoshi, Mobile phone base station radiation does not affect neoplastic transformation in BALB/3T3 cells, *Bioelectromagnetics* 29 (2008) 55–64.
- [30] G. Neubauer, M. Feychting, Y. Hamnerius, L. Kheifets, N. Kuster, I. Ruiz, J. Schüz, R. Überbacher, J. Wiart, Feasibility of future epidemiological studies on possible health effects of mobile phone base stations, *Bioelectromagnetics* 28 (2007) 224–230.
- [31] P. Kaatsch, C. Spix, R. Schulze-Rath, S. Schmiedel, M. Blettner, Leukaemia in young children living in the vicinity of German nuclear power plants, *Int. J. Cancer* 122 (2008) 721–726.





## Review

# Long-term exposure to magnetic fields and the risks of Alzheimer's disease and breast cancer: Further biological research

Zoreh Davanipour<sup>a,\*</sup>, Eugene Sobel<sup>b</sup>

<sup>a</sup> Northwestern University, Feinberg School of Medicine, Chicago, IL, United States

<sup>b</sup> Friends Research Institute, Los Angeles, CA, United States

Received 1 November 2008; accepted 30 January 2009

## Abstract

**Objective:** Extremely low frequency (ELF) and radio frequency (RF) magnetic fields (MFs) pervade our environment. Whether or not these magnetic fields are associated with increased risk of serious diseases, e.g., cancers and Alzheimer's disease, is thus important when developing a rational public policy. The Bioinitiative Report was an effort by internationally recognized scientists who have spent significant time investigating the biological consequences of exposures to these magnetic fields to address this question. Our objective was to provide an unbiased review of the current knowledge and to provide our general and specific conclusions. **Results:** The evidence indicates that long-term significant occupational exposure to ELF MF may certainly increase the risk of both Alzheimer's disease and breast cancer. There is now evidence that two relevant biological processes (increased production of amyloid beta and decreased production of melatonin) are influenced by high long-term ELF MF exposure that may lead to Alzheimer's disease. There is further evidence that one of these biological processes (decreased melatonin production) may also lead to breast cancer. Finally, there is evidence that exposures to RF MF and ELF MF have similar biological consequences. **Conclusion:** It is important to mitigate ELF and RF MF exposures through equipment design changes and environmental placement of electrical equipment, e.g., AC/DC transformers. Further research related to these proposed and other biological processes is required.

© 2009 Elsevier Ireland Ltd. All rights reserved.

**Keywords:** Extremely low frequency (ELF); Magnetic fields (MFs); Amyloid beta (A $\beta$ ); Melatonin; Alzheimer's disease (AD)

## Contents

1. Introduction .....	00
2. Epidemiologic studies presented in the Bioinitiative Report related to Alzheimer's disease and breast cancer .....	00
2.1. Alzheimer's disease .....	00
2.2. Breast cancer .....	00
3. Biological hypotheses relating ELF MF exposure to Alzheimer's disease and breast cancer .....	00
3.1. ELF MF exposure and peripheral and brain production of amyloid beta (Fig. 1) .....	00
3.2. Melatonin—background .....	00
3.3. Low melatonin production and Alzheimer's disease .....	00
3.4. Low melatonin production and breast cancer .....	00
4. Discussion and future research .....	00
4.1. Epidemiologic studies .....	00
4.2. ELF and/or RF MF exposure mitigation .....	00
4.3. Biological mechanisms/experimental medicine research .....	00
Acknowledgement .....	00
References .....	00

\* Corresponding author.

E-mail address: zdavani@earthlink.net (Z. Davanipour).

## 1. Introduction

In this review, we emphasize (a) two proposed biological models “explaining” the apparent relationship between high, long-term exposure to extremely low frequency (ELF) magnetic fields (MFs) and Alzheimer’s disease (AD), one of which also relates to breast cancer and (b) areas of biological research needed to confirm or refute these models. Prior to this discussion, we provide the conclusions from our detailed review chapter (Section 12: Davanipour and Sobel [1]) in the Bioinitiative Report [2] related to epidemiologic research, which initially identified these relationships. We refer the reader to Section 12 and supporting, peer-reviewed papers for details of the epidemiologic studies discussed in that section. Other papers in this issue of Pathophysiology (e.g., on the stress response and DNA strand breaks) demonstrate that exposures to ELF MF and radio frequency (RF) MF often have the same biological consequences.

## 2. Epidemiologic studies presented in the Bioinitiative Report related to Alzheimer’s disease and breast cancer

The conclusions reached from our detailed review of the literature in Section 12 in the Bioinitiative Report (see references for URL) on long-term significant ELF MF exposure and Alzheimer’s disease and breast cancer are provided below [1]. The section references below refer to sub-sections of Section 12 of the Bioinitiative Report.

**Melatonin production (Section II).** Eleven of the 13 published epidemiologic residential and occupational studies are considered to provide (positive) evidence that high long-term ELF MF exposure can result in decreased melatonin production. The two negative studies had important deficiencies which may certainly have biased the results. Thus, there is sufficient evidence to conclude that long-term relatively high ELF MF exposure can result in a decrease in melatonin production. It has not been determined to what extent personal characteristics, e.g., medications, interact with ELF MF exposure in decreasing melatonin production.

### 2.1. Alzheimer’s disease

Section 12 of the Bioinitiative Report provides the details of the following conclusions.

- There is initial evidence that (i) a high level of peripheral amyloid beta, generally considered the primary neurotoxic agent when aggregated, is a risk factor for AD and (ii) medium to high MF exposure can increase peripheral amyloid beta. High brain levels of amyloid beta are also a risk factor for AD and medium to high MF exposure to brain cells likely also increases these cells’ production of amyloid beta (Section IIIA).

- There is considerable *in vitro* and animal evidence that melatonin protects against AD. Therefore, it is certainly possible that low levels of melatonin production are associated with an increase in the risk of AD (Section IIIB).
- There is strong epidemiologic evidence that long-term exposure to ELF MF is a risk factor for AD. There are seven studies of ELF MF exposure and AD that met our inclusion criteria. Six of these studies are more of less positive and only one is negative. The negative study has a serious deficiency in ELF MF exposure classification which results in subjects with rather low exposure being considered as having significant exposure. Several published studies were excluded from further consideration due to serious deficiencies, primarily diagnostic inaccuracy (e.g., use of death certificates for diagnosis of AD) and/or serious exposure assessment problems. These latter studies likely had risk estimated seriously biased towards the null hypothesis of no risk. It should be noted, however, that even some of these studies were positive (Sections IIIC and IIID).

### 2.2. Breast cancer

There is sufficient evidence from *in vitro* and animal studies, from human biomarker studies, from occupational and light at night case-control studies, and the only two longitudinal studies with appropriate collection of urine samples to conclude that high ELF MF exposure may certainly be a risk factor for breast cancer (Section IV). Note that at the time the Bioinitiative Report was made public, there was only one longitudinal study with appropriate collection of urine samples. There are now two such studies [3,4].

**Seamstresses.** Seamstress is, in fact, one of the most highly ELF MF exposed occupations, with exposure levels generally well above 10mG over a significant proportion of the workday. Seamstresses have been consistently found to be at higher risk of Alzheimer’s disease and breast cancer. This occupation deserves specific attention in future studies. We are unaware of any measurements of RF MF among seamstresses (Section V and throughout Section 12).

## 3. Biological hypotheses relating ELF MF exposure to Alzheimer’s disease and breast cancer

Two biological hypotheses are discussed. The first one relates ELF MF exposure to increased amyloid beta ( $A\beta$ ) production and subsequent development of AD. The second one relates ELF MF exposure to decreased melatonin production. Decreased melatonin production appears to have differing deleterious consequences related to AD and breast cancer development.

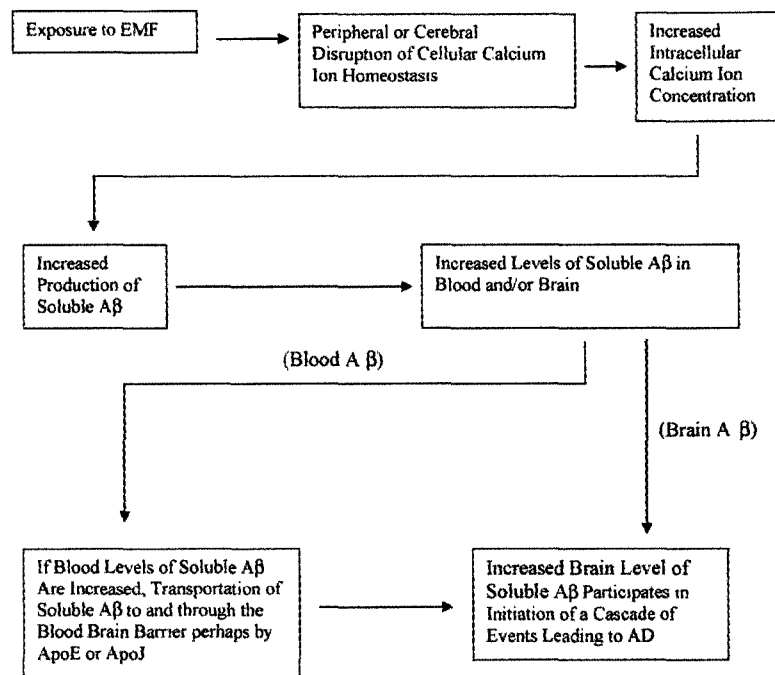


Fig. 1. Hypothesized biological pathway from MF exposure to AD Development (from Sobel and Davanipour [5]).

### 3.1. ELF MF exposure and peripheral and brain production of amyloid beta (Fig. 1)

The ELF MF exposure and increased amyloid beta hypothesis was developed by Sobel and Davanipour as a result of our initial findings that long-term ELF MF occupational exposure was a risk factor for AD [5] (see Fig. 1). Seamstress was the most common occupation among subjects with AD in the five databases we investigated [6–8]. ELF MF exposure among seamstresses had not been measured prior to our 1995 study [6]. Beginning in 1994, we measured a very large number of seamstresses working in either a factory setting or individually. Their exposures were very high, particularly when using an industrial sewing machine. The highest exposures were, however, not to the brain, because the motor on industrial machines is located at the knees. The motor or AC/DC transformer in home sewing machines is in the machine arm located near the operator's chest and right arm. This peripheral exposure led us to consider how peripheral ELF MF exposure might be associated with development of amyloid plaques in the brain.

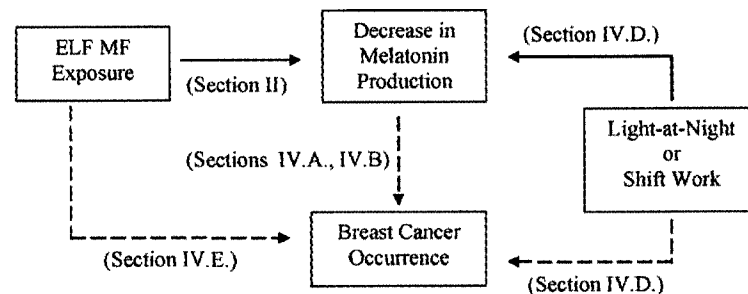
Our biologically plausible hypothesis relating MF exposure to AD is based on the independent work of many researchers in several different fields. Details and references are provided in Sobel and Davanipour [5]. Briefly, the hypothesized process involves increased peripheral or brain production of Aβ as a result of MF exposure causing voltage-gated calcium ion channels to be open longer than normal. This results in abnormally high intracellular levels of calcium ions which in turn results in the production of Aβ. The result-

ing Aβ is quickly secreted into the blood. If peripheral, the Aβ is then transported to and through the blood–brain barrier, perhaps best chaperoned by the ε4 isoform of apolipoprotein E (apoE). (Note that this might be one reason why the ε4 isoform is a risk factor for AD.) Fig. 1 provides a schematic outline of the hypothesis. Each step in the proposed pathway is supported by *in vitro* studies.

At the time of publication of this hypothesis, no human studies related to this hypothesis had been conducted. There are now two groups that have published relevant studies, without apparently any knowledge of our hypothesis—or at least no reference to the hypothesis: (1) high levels of peripheral Aβ<sub>1–42</sub>, the more neurotoxic version of Aβ, has been found to be a risk factor for AD [9,10]; acute exposure to ELF MF increases peripheral Aβ [11]. Details may also be found in the Bioinitiative Report (Section IIIA) [1].

### 3.2. Melatonin—background

Melatonin is found in every cell of the body and readily crosses the blood–brain barrier. It scavenges reactive oxygen species (ROS) at both physiologic and pharmacologic concentrations. In the literature, “physiologic” refers to blood level concentrations of melatonin, while “pharmacologic” indicates 2–3 orders of magnitude higher concentration. Recently, intracellular levels of melatonin, especially within the nucleus, have been shown to be naturally at “pharmacologic” levels for all cellular organelles studied to date [12,13].



Note: Dashed lines indicate studies directly relating ELF MF exposure, light-at-night or shift work, or lower melatonin production to breast cancer occurrence. Section references refer to Section 12 of the Bioinitiative Report [1].

Fig. 2. Outline of the evidence that ELF MF exposure causes breast cancer through decreases in melatonin production—with section references to Section 12, Bioinitiative Report [1]. Note: Dashed lines indicate studies directly relating ELF MF exposure, light-at-night or shift work, or lower melatonin production to breast cancer occurrence.

### 3.3. Low melatonin production and Alzheimer's disease

Numerous *in vitro* and animal studies indicate that melatonin may be *protective* against AD and thus low or lowered melatonin production may be a risk factor for AD. These studies have found that melatonin has the following effects:

- Inhibition of the neurotoxicity and cytotoxicity of A $\beta$ , including in mitochondria [14–19];
- Inhibition of the formation of  $\beta$ -pleated sheet structures and A $\beta$  fibrils [20–25];
- Reversal of the profibrillogenic activity of apolipoprotein E  $\epsilon$ 4, an isoform conferring increased risk of AD [21];
- Inhibition of the oxidative stress *in vitro* and in transgenic mouse models of AD, if given early [23,26,27], but not necessarily if given to old mice [28];
- Increase in survival time in mouse models of AD [23];
- Reduction of oxidative stress and of proinflammatory cytokines induced by A $\beta$ <sub>1–40</sub> in rat brain *in vitro* and *in vivo* [29–31];
- Decrease of the prevalence of A $\beta$ <sub>1–40</sub> and A $\beta$ <sub>1–42</sub> in the brain in young and middle aged mice [32];
- Improvement of memory and learning in rat models of AD pathology [33,34], but not necessarily in A $\beta$ -infused rat models [35].

Note that transgenic mouse models of AD mimic senile plaque accumulation, neuronal loss, and memory impairment. There have been several reviews, e.g., [36–39]. Thus, chronic low levels of melatonin production may be etiologically related to AD incidence [40].

### 3.4. Low melatonin production and breast cancer

See Fig. 2 for a diagram of the discussed relationships between ELF MF exposure and breast cancer risk.

*In vitro* studies related to prevention of oxidative damage. Well over 1000 publications have found that melatonin neu-

tralizes hydroxyl radicals and reduces oxidative damage. For reviews see Tan et al. [41] and Peyrot and Ducrocq [42]. Melatonin has also been shown to act synergistically with vitamin C, vitamin E and glutathione [43] and stimulates the antioxidant enzymes superoxide dismutase, glutathione peroxidase and glutathione reductase [44]. Furthermore,

- melatonin neutralizes hydroxyl radicals more efficiently than does reduced glutathione [45,46];
- melatonin reduces oxidative damage to macromolecules in the presence of free radicals [47,48] due at least to its free radical scavenging properties [49];
- melatonin increases the effectiveness of other antioxidants, e.g., superoxide dismutase, glutathione peroxidase, and catalase [50–54];
- melatonin has protective effects against ultraviolet and ionizing radiation [55–57];
- melatonin has been found to be a more potent protector from oxidative injury than vitamin C or vitamin E (micro-moles/kg) (for a review of the evidence, see: Tan et al. [43];
- melatonin was also found *in vitro* to scavenge peroxy radicals more effectively than vitamin E, vitamin C or reduced glutathione [58], although melatonin is not a very strong scavenger of peroxy radicals [49].

*Animal studies of melatonin and mammary tumor prevention.* Several studies have found that melatonin inhibits the incidence of mammary tumors in laboratory animals either prone to such tumors or exposed to a carcinogen (e.g., [50–63]). Tan et al. [64,65] found that melatonin at both physiological and pharmacological levels protected Sprague–Dawley rats from safrole induced liver DNA adduct formation. Melatonin and retinoic acid appear to act synergistically in the chemoprevention of animal model tumors [66] and *in vitro* systems [67].

*Melatonin prevents oxidative DNA damage by estradiol and radiation.* Karbownik et al. [68] found that melatonin

protects against DNA damage in the liver and kidney of male hamsters caused by estradiol treatment. Several studies have found that laboratory animals are protected by melatonin from lethal doses of ionizing radiation (e.g., [69–71]). Vijayalaxmi et al. [70] and Karbownik et al. [71] also investigated markers of oxidative DNA damage and found significant decreases in these markers in the melatonin treated animals.

**Melatonin: Scavenger of  $\bullet\text{OH}$  and Other ROS.** Melatonin is a powerful, endogenously produced scavenger of reactive oxygen species (ROS), particularly the hydroxyl radical ( $\bullet\text{OH}$ ). Other ROS which melatonin scavenges include hydrogen peroxide ( $\text{H}_2\text{O}_2$ ), nitric oxide ( $\text{NO}\bullet$ ), peroxy-nitrite anion ( $\text{ONOO}^-$ ), hypochlorous acid ( $\text{HOCl}$ ), and singlet oxygen ( $^1\text{O}_2$ ) [50,72–75].  $\bullet\text{OH}$  is produced at high levels by natural aerobic activity. ROS are also produced by various biological activities or result from certain environmental and lifestyle (e.g., smoking) exposures.  $\bullet\text{OH}$  is the most reactive and cytotoxic of the ROS [76].  $\bullet\text{OH}$  appears not to be removed by antioxidative enzymes, but is only detoxified by certain direct radical scavengers such as melatonin [77].

#### 4. Discussion and future research

Other papers in this special issue of Pathophysiology provide evidence that RF MF exposure and ELF MF exposure may have similar biological consequences.

We primarily limit our discussion of future research to studies in humans with experimental medicine components, emphasizing the latter. However, we initially discuss limiting exposures.

It should be noted that ELF MF exposure may also be associated with other cancers. This may be because of the decrease in melatonin production and melatonin's varying antioxidant, anti-inflammation, and immune response enhancement properties.

##### 4.1. Epidemiologic studies

The incidence rates of Alzheimer's disease and breast cancer are increasing. These increases are certainly in part due to our living longer, at least for AD, if not better lives. However, environmental exposures are likely to play important roles. At the same time, ELF and RF MF exposure is becoming more and more common in our world. In our three published studies of MF and AD, approximately 7.4–12.0% of the cases and 3.4–5.3% of the controls had primary occupations associated with medium or high ELF MF exposure [6–8]. Many more subjects may have had exposures from sources generally not identified in epidemiologic studies, because individualized 'on-site' exposure assessment is usually not feasible. We give two examples coming from 'onsite' inspections we have performed: a subject who had developed amyotrophic lateral sclerosis (ALS) had spent many years with a 75 mG ELF MF exposure due to having his foot on

a deadbolt lock/unlock foot device for his office door under his desk; a subject who had developed AD who spent over 25 years sitting at his home desk for at least 4 h per day in a chair backed up to a wall with a fuse box directly on the other side of the wall which produced a very high ELF MF exposure to his back and head. (Note that there is also significant epidemiologic evidence that ELF MF exposure is a risk factor for ALS.) The frequencies of such exposures in studies are unknown.

As is often the case, more research is required. However, the designs of this future research should be informed and directed by the results of previous research. Future epidemiologic studies should use subjects for whom it is unequivocally known that the ELF MF and/or RF MF exposure is high and matched subjects for whom such exposure is known to be low. Matching criteria should include age, gender, and residential environment so as to at least partially exclude other exposures.

There should be additional studies related to the levels of production of peripheral amyloid beta, particularly  $\text{A}\beta_{1-42}$ , and melatonin, on the one hand, and both MF exposure and the risk of AD, on the other hand. Such studies need to be able to investigate the possible associations between peripheral amyloid beta and melatonin levels and both earlier/concurrent MF exposure and subsequent development of AD. Similar studies need to be carried out for breast cancer, excluding the amyloid beta component. This effort will likely require both retrospective and longitudinal studies. There are only two known longitudinal studies [3,4] which collected urine samples at baseline so that overnight pre-morbid melatonin production was reliably estimated. These studies found an association between low melatonin production and breast cancer. These studies may also be able to provide important additional information if it is possible to determine MF exposures with reasonable accuracy and follow-up AD status on a sufficient number of participants.

Case-control studies of melatonin as a risk factor for AD and breast cancer are hampered by the fact that biological sequelae of both AD and breast cancer result in a decline of melatonin production to an unknown extent. (In breast cancer patients, there is a melatonin production rebound when tumors are surgically removed. In AD patients, the production of serotonin, the precursor of melatonin, is decreased and noradrenergic regulation becomes dysfunctional [78].) However, melatonin production is partially under genetic control. We have conducted a study of relatively healthy members of nuclear families and melatonin production (DOD Congressionally Directed Medical Research Program Grant: DAMD17-00-1-0692). The production of melatonin of the mother was successfully modelled as a function of the melatonin of a daughter, after adjusting for both the daughter's age and the influence of the father. This work allows for the design of case-control studies of the influence of long-term MF exposure on both melatonin production and the risks of breast cancer and AD.

#### 4.2. ELF and/or RF MF exposure mitigation

It is also vital to mitigate both the extent of MF exposure and the effects of such exposure. Mitigation means efforts to both locate and shield or move the sources of MF away from individuals and design equipment which produces lower levels of MF. Little effort has apparently been spent on design issues. There are simple things that can be done. For example, almost all AC/DC transformers emit about 75 mG ELF MF fields. The exception, in our experience, has been a few transformers for Apple laptops measured about 10 years ago. AC/DC transformers are now everywhere, specifically under and around office desks and in nearly every room in a residence, often near the heads of beds. Maximizing one's distance from a transformer is important, because the strength of the MF field drops off with the square or cube of the distance from the source.

Seamstress is a very common profession and being a seamstress is clearly a risk factor for AD and quite possibly for breast cancer also. Seamstresses experience higher ELF MF exposure than members of almost any other profession. Older industrial sewing machines are extremely common all over the world. They produce extremely strong MFs, but it is possible to design "covers" for the motor to interfere with these fields, much as "headphones" can mitigate sound waves. Newer computer driven home sewing machines produce MF because of the AC/DC transformer. These transformers are placed in the arm of the machine, which results in high MF exposure to the operator. Simply by connecting the transformer to the machine by an electrical cord about three or more feet from the operator would mitigate a significant percentage of the MF exposure.

#### 4.3. Biological mechanisms/experimental medicine research

We argue that, to the extent possible, research should now be conducted in humans. We list the following research questions as important examples of studying the biological effects of ELF and/or RF MF exposure:

1. Generation of peripheral amyloid beta
  - a. Determination of intracellular  $\text{Ca}^{2+}$  ion concentration changes as a consequence of ELF or RF MF exposure.
  - b. Measurement of the amount of  $\text{A}\beta_{1-42}$  and  $\text{A}\beta_{1-40}$  produced by and secreted from cells.
    - i. This could be done at least by measuring blood levels of amyloid before and after ELF and/or RF MF exposure.
    - ii. Perhaps there are more sophisticated experimental designs.
  - c. Determination of which cell types in fact produce more amyloid beta after or during ELF and/or RF MF exposure.
  - d. Determination of the dose response relationship(s) between ELF and/or RF MF exposure and cellular amyloid beta production.

- e. Measurement of the accumulation of amyloid beta in the brain, perhaps using PET scans [79,80].

#### 2. Decrease in melatonin production

*Note:* it is known that the pineal gland, the primary source of melatonin, has a tendency to become calcified and, perhaps, this is the reason why generally there is a reduction of melatonin production during aging.

- a. Determination of the extent of intracellular calcium within the pineal gland as a result of acute ELF and/or RF MF exposure.
- b. Determination of the extent of calcification of the pineal gland as a result of varying levels of long-term ELF and/or RF MF exposure.

#### Acknowledgement

The authors would like to thank Dr. James Burch, Arnold School of Public Health, University of South Carolina, for his review of Section 12 in the Bioinitiative Report.

#### References

- [1] Z. Davanipour, E. Sobel, Section 12: Magnetic Field Exposure: Melatonin Production; Alzheimer's Disease; Breast Cancer, in: C.F. Blackman, M. Blank, M. Kundi, C. Sage (Organizing Committee), BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF), 2007. URL: [http://www.bioinitiative.org/report/docs/section\\_12.pdf](http://www.bioinitiative.org/report/docs/section_12.pdf).
- [2] BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF), 2007. URL: <http://www.bioinitiative.org/report/index.htm>.
- [3] E.S. Schernhammer, S.E. Hankinson, Urinary melatonin levels and breast cancer risk, *J. Natl. Cancer Inst.* 97 (2005) 1084–1087.
- [4] E.S. Schernhammer, F. Berrino, V. Krogh, G. Secreto, A. Micheli, E. Venturelli, S. Sieri, C.T. Sempos, A. Cavalleri, H.J. Schünemann, S. Strano, P. Muti, Urinary 6-sulfatoxymelatonin levels and risk of breast cancer in postmenopausal women, *J. Natl. Cancer Inst.* 100 (2008) 898–905.
- [5] E. Sobel, Z. Davanipour, Electromagnetic field exposure may cause increased production of amyloid beta and eventually lead to Alzheimer's disease, *Neurology* 47 (1996) 1594–1600.
- [6] E. Sobel, Z. Davanipour, R. Sulkava, T. Erkinjuntti, J. Wikstrom, V.W. Henderson, G. Buckwalter, J.D. Bowman, P.J. Lee, Occupations with exposure to electromagnetic fields: a possible risk factor for Alzheimer's disease, *Am. J. Epidemiol.* 142 (1995) 515–524.
- [7] E. Sobel, M. Dunn, Z. Davanipour, Z. Qian, H.C. Chui, Elevated risk of Alzheimer's disease among workers with likely electromagnetic field exposure, *Neurology* 47 (1996) 1477–1481.
- [8] Z. Davanipour, C.-C. Tseng, P.-J. Lee, E. Sobel, A case-control study of occupational magnetic field exposure and Alzheimer's disease: results from the California Alzheimer's Disease Diagnosis and Treatment Centers, *BMC Neurol.* 7 (2007) 13, <http://www.biomedcentral.com/1471-2377/7/13>.
- [9] R. Mayeux, W.X. Tang, D.M. Jacobs, J. Manly, K. Bell, C. Merchant, S.A. Small, Y. Stern, H.M. Wisniewski, P.D. Mehta, Plasma amyloid beta-peptide 1–42 and incipient Alzheimer's disease, *Ann. Neurol.* 46 (1999) 412–416.
- [10] R. Mayeux, L.S. Honig, M.X. Tang, J. Manly, Y. Stern, N. Schupf, P.D. Mehta, Plasma A[ $\beta$ 40] and A[ $\beta$ 42] and Alzheimer's disease: relation to age, mortality, and risk, *Neurology* 61 (2003) 1185–1190.



- [11] C. Noonan, J. Reif, J. Burch, T.Y. Ichinose, M.G. Yost, K. K. Magnusson, Relationship between amyloid  $\beta$  protein and melatonin metabolite in a study of electric utility workers, *J. Occup. Environ. Med.* 44 (2002) 769–775.
- [12] G.J.M. Maestroni, Therapeutic potential of melatonin in immunodeficiency states, viral diseases, and cancer, in: G. Heuther, W. Kochen, T.J. Simat, H. Steinhart (Eds.), *Tryptophan, Serotonin, and Melatonin: Basic Aspects and Applications*, Kluwer Academic/Plenum Publishers, New York, 1999, pp. 217–226.
- [13] R.J. Reiter, D.-X. Tan, W. Qi, L.C. Manchester, M. Karbownik, J.R. Calvo, Pharmacology and physiology of melatonin in the reduction of oxidative stress in vivo, *Biol. Signals Recept.* 9 (2000) 160–171.
- [14] M.A. Pappolla, M. Sos, R.A. Omar, R.J. Bick, D.L. Hickson-Bick, R.J. Reiter, S. Efthimiopoulos, N.K. Robakis, Melatonin prevents death of neuroblastoma cells exposed to the Alzheimer amyloid peptide, *J. Neurosci.* 17 (1997) 1683–1690.
- [15] M.A. Pappolla, Y.J. Chyan, B. Poeggeler, P. Bozner, J. Ghiso, S.P. LeDoux, G.L. Wilson, Alzheimer beta protein mediated oxidative damage of mitochondrial DNA: prevention by melatonin, *J. Pineal Res.* 27 (1999) 226–229.
- [16] M.A. Pappolla, M.J. Simovich, T. Bryant-Thomas, Y.J. Chyan, B. Poeggeler, M. Dubocovich, R. Bick, G. Perry, F. Cruz-Sanchez, M.A. Smith, The neuroprotective activities of melatonin against the Alzheimer beta-protein are not mediated by melatonin membrane receptors, *J. Pineal Res.* 32 (2002) 135–142.
- [17] Y.X. Shen, W. Wei, S.Y. Xu, Protective effects of melatonin on cortico-hippocampal neurotoxicity induced by amyloid beta-peptide 25–35, *Acta Pharmacol. Sin.* 23 (2002) 71–76.
- [18] P. Zatta, G. Tognon, P. Carampin, Melatonin prevents free radical formation due to the interaction between beta-amyloid peptides and metal ions [Al(III), Zn(II), Cu(II), Mn(II), Fe(II)], *J. Pineal Res.* 35 (2003) 98–103.
- [19] M.H. Jang, S.B. Jung, M.H. Lee, C.J. Kim, Y.T. Oh, I. Kang, J. Kim, E.H. Kim, Melatonin attenuates amyloid beta25–35-induced apoptosis in mouse microglial BV2 cells, *Neurosci. Lett.* 380 (2005) 26–31.
- [20] M. Pappolla, P. Bozner, C. Soto, H. Shao, N.K. Robakis, M. Zagorski, B. Frangione, J. Ghiso, Inhibition of Alzheimer beta-fibrillogenesis by melatonin, *J. Biol. Chem.* 273 (1998) 7185–7188.
- [21] B. Poeggeler, L. Miravalle, M.G. Zagorski, T. Wisniewski, Y.J. Chyan, Y. Zhang, H. Shao, T. Bryant-Thomas, R. Vidal, B. Frangione, J. Ghiso, M.A. Pappolla, Melatonin reverses the profibrillogenic activity of apolipoprotein E4 on the Alzheimer amyloid A $\beta$  peptide, *Biochemistry* 40 (2001) 14995–15001.
- [22] Z. Skribanek, L. Balaspiri, M. Mak, Interaction between synthetic amyloid-beta-peptide (1–40) and its aggregation inhibitors studied by electrospray ionization mass spectrometry, *J. Mass Spectrom.* 36 (2001) 1226–1229.
- [23] E. Matsubara, T. Bryant-Thomas, J. Pacheco Quinto, T.L. Henry, B. Poeggeler, D. Herbert, F. Cruz-Sanchez, Y.J. Chyan, M.A. Smith, G. Perry, M. Shoji, K. Abe, A. Leone, I. Grundke-Iqbal, G.L. Wilson, J. Ghiso, C. Williams, L.M. Refolo, M.A. Pappolla, D.G. Chain, E. Neria, Melatonin increases survival and inhibits oxidative and amyloid pathology in a transgenic model of Alzheimer's disease, *J. Neurochem.* 85 (2003) 1101–1108 (Erratum in: *J. Neurochem.* 86 (2003) 1312).
- [24] Z. Feng, Y. Chang, Y. Cheng, B.L. Zhang, Z.W. Qu, C. Qin, J.T. Zhang, Melatonin alleviates behavioral deficits associated with apoptosis and cholinergic system dysfunction in the APP 695 transgenic mouse model of Alzheimer's disease, *J. Pineal Res.* 37 (2004) 129–136.
- [25] X. Cheng, R.B. van Breemen, Mass spectrometry-based screening for inhibitors of  $\beta$ -amyloid protein aggregation, *Anal. Chem.* 77 (2005) 7012–7015.
- [26] K.L. Clapp-Lilly, M.A. Smith, G. Perry, P.L. Harris, X. Zhu, L.K. Duffy, Melatonin acts as antioxidant and pro-oxidant in an organotypic slice culture model of Alzheimer's disease, *Neuroreport* 12 (2001) 1277–1280.
- [27] Z. Feng, C. Qin, Y. Chang, J.T. Zhang, Early melatonin supplementation alleviates oxidative stress in a transgenic mouse model of Alzheimer's disease, *Free Radic. Biol. Med.* 40 (2006) 101–109.
- [28] J. Quinn, D. Kulhanek, J. Nowlin, R. Jones, D. Pratico, J. Rokach, R. Stackman, Chronic melatonin therapy fails to alter amyloid burden or oxidative damage in old Tg2576 mice: implications for clinical trials, *Brain Res.* 1037 (2005) 209–213.
- [29] K.L. Clapp-Lilly, M.A. Smith, G. Perry, L.K. Duffy, Melatonin reduces interleukin secretion in amyloid-beta stressed mouse brain slices, *Chem. Biol. Interact.* 134 (2001) 101–107.
- [30] Y.X. Shen, S.Y. Xu, W. Wei, X.X. Sun, L.H. Liu, J. Yang, C. Dong, The protective effects of melatonin from oxidative damage induced by amyloid beta-peptide 25–35 in middle-aged rats, *J. Pineal Res.* 32 (2002) 85–89.
- [31] S. Rosales-Corral, D.-X. Tan, R.J. Reiter, M. Valdivia-Velazquez, G. Martinez-Barboza, J.P. Acosta-Martinez, G.G. Ortiz, Orally administered melatonin reduces oxidative stress and proinflammatory cytokines induced by amyloid-beta peptide in rat brain: a comparative, in vivo study versus vitamin C and E, *J. Pineal Res.* 35 (2003) 80–84.
- [32] D.K. Lahiri, D. Chen, Y.W. Ge, S.C. Bondy, E.H. Sharman, Dietary supplementation with melatonin reduces levels of amyloid beta-peptides in the murine cerebral cortex, *J. Pineal Res.* 36 (2004) 224–231.
- [33] Y.X. Shen, W. Wei, J. Yang, C. Liu, C. Dong, S.Y. Xu, Improvement of melatonin to the learning and memory impairment induced by amyloid beta-peptide 25–35 in elder rats, *Acta Pharmacol. Sin.* 22 (2001) 797–803.
- [34] M. Weinstock, S. Shoham, Rat models of dementia based on reductions in regional glucose metabolism, cerebral blood flow and cytochrome oxidase activity, *J. Neural Transm.* 111 (2004) 347–366.
- [35] F. Tang, S. Nag, S.Y. Shiu, S.F. Pang, The effects of melatonin and Ginkgo biloba extract on memory loss and choline acetyltransferase activities in the brain of rats infused intracerebroventricularly with beta-amyloid 1–40, *Life Sci.* 71 (2002) 2625–2631.
- [36] D.P. Cardinali, A.M. Furio, M.P. Reyes, Clinical perspectives for the use of melatonin as a chronobiotic and cytoprotective agent, *Ann. NY Acad. Sci.* 1057 (2005) 327–336.
- [37] V. Srinivasan, S. Pandi-Perumal, D. Cardinali, B. Poeggeler, R. Hardeland, Melatonin in Alzheimer's disease and other neurodegenerative disorders, *Behav. Brain Funct.* 2 (2006) 15, doi:10.1186/1744-9081-2-15.
- [38] Y. Cheng, Z. Feng, Q.Z. Zhang, J.T. Zhang, Beneficial effects of melatonin in experimental models of Alzheimer disease, *Acta Pharmacol. Sin.* 27 (2006) 129–139.
- [39] J.Z. Wang, Z.F. Wang, Role of melatonin in Alzheimer-like neurodegeneration, *Acta Pharmacol. Sin.* 27 (2006) 41–49.
- [40] Y.H. Wu, D.F. Swaab, The human pineal gland and melatonin in aging and Alzheimer's disease, *J. Pineal Res.* 38 (2005) 145–152.
- [41] D.-X. Tan, L.C. Manchester, M.P. Terron, L.J. Flores, R.J. Reiter, One molecule, many derivatives: a never-ending interaction of melatonin with reactive oxygen and nitrogen species? *J. Pineal Res.* 42 (2007) 28–42.
- [42] F. Peyrot, C. Ducrocq, Potential role of tryptophan derivatives in stress responses characterized by the generation of reactive oxygen and nitrogen species, *J. Pineal Res.* 45 (2008) 235–246.
- [43] D.-X. Tan, L.C. Manchester, R.J. Reiter, W.B. Qi, M. Karbownik, J.R. Calvo, Significance of melatonin in antioxidative defense system: reactions and products, *Biol. Signals Recept.* 9 (2000) 137–159.
- [44] R.J. Reiter, D.-X. Tan, S. Burkhardt, Reactive oxygen and nitrogen species and cellular and organismal decline: amelioration with melatonin, *Mech. Ageing Dev.* 123 (2002) 1007–1019.
- [45] D.-X. Tan, L.D. Chen, B. Poeggeler, L.C. Manchester, R.J. Reiter, Melatonin: a potent, endogenous hydroxyl radical scavenger, *Endocr. J.* 1 (1993) 57–60.
- [46] H.J. Bromme, W. Morke, D. Peschke, H. Ebel, D. Peschke, Scavenging effect of melatonin on hydroxyl radicals generated by alloxan, *J. Pineal Res.* 29 (2000) 201–208.

- [47] R.J. Reiter, L. Tang, J.J. Garcia, A. Munoz-Hoyos, Pharmacological actions of melatonin in oxygen radical pathophysiology, *Life Sci.* 60 (1997) 2255–2271.
- [48] R.J. Reiter, D.-X. Tan, L.C. Manchester, W. Qi, Biochemical reactivity of melatonin with reactive oxygen and nitrogen species: a review of the evidence, *Cell Biochem. Biophys.* 34 (2001) 237–256.
- [49] R.J. Reiter, D. Acuna-Castroviejo, D.-X. Tan, S. Burkhardt, Free radical-mediated molecular damage: mechanisms for the protective actions of melatonin in the central nervous system, *Ann. NY Acad. Sci.* 939 (2001) 200–215.
- [50] I. Antolin, B. Obst, S. Burkhardt, R. Hardeland, Antioxidative protection in a high-melatonin organism: The dinoflagellate *Gonyaulax polydora* is rescued from lethal oxidative stress by strongly elevated, but physiologically possible, concentrations of melatonin, *J. Pineal Res.* 23 (1997) 182–190.
- [51] M. Kotler, C. Rodriguez, R.M. Sainz, I. Antolin, A. Menendez-Pelaez, Melatonin increases gene expression for antioxidant enzymes in rat brain cortex, *J. Pineal Res.* 24 (1998) 83–89.
- [52] M.I. Pablos, J.I. Chuang, J.R. Reiter, G.G. Ortiz, W.M. Daniels, E. Sewerynek, D. Melchiorri, B. Poeggeler, Time course of melatonin-induced increase in glutathione peroxidase activity in chick tissues, *Biol. Signals* 4 (1995) 325–330.
- [53] L.R. Barlow-Walden, R.J. Reiter, A. Abe, M. Pablos, A. Menendez-Pelaez, L.D. Chen, B. Poeggeler, Melatonin stimulated brain glutathione peroxidase activity, *Neurochem. Int.* 26 (1995) 497–502.
- [54] P. Montilla, I. Tunes, M.C. Munoz, J.V. Soria, A. Lopez, Antioxidative effect of melatonin in rat brain oxidative stress induced by Adriamycin, *Rev. Esp. Fisiol.* 53 (1997) 301–305.
- [55] Vijayalaxmi, R.J. Reiter, M.L. Meltz, Melatonin protects human blood lymphocytes from radiation-induced chromosome damage, *Mutat. Res.* 346 (1995) 23–31.
- [56] Vijayalaxmi, R.J. Reiter, T.S. Herman, M.L. Meltz, Melatonin and radioprotection from genetic damage: DNA damage in human blood lymphocytes, *Mutat. Res.* 371 (1996) 221–228.
- [57] Vijayalaxmi, R.J. Reiter, M.L. Meltz, T.S. Herman, Melatonin: possible mechanisms involved in its “radioprotective” effect, *Mutat. Res.* 404 (1998) 187–189.
- [58] C. Pieri, M. Marra, F. Moroni, R. Recchioni, F. Marcheselli, Melatonin: a peroxyl radical scavenger more effective than vitamin E, *Life Sci.* 55 (1994) 271–276.
- [59] L. Tamarkin, M. Cohen, D. Roselle, C. Reichert, M. Lippman, B. Chabner, Melatonin inhibition and pinealectomy enhancement of 7,12-dimethylbenz(a)anthracene-induced mammary tumors in the rat, *Cancer Res.* 41 (1981) 4432–4436.
- [60] P.N. Shah, M.C. Mhatre, L.S. Kothari, Effect of melatonin on mammary carcinogenesis in intact and pinealectomized rats in varying photoperiods, *Cancer Res.* 44 (1984) 3403–3407.
- [61] L.S. Kothari, P.N. Shah, M.C. Mhatre, Pineal ablation in varying photoperiods and the incidence of 9,10-dimethyl-1,2-benzanthracene induced mammary cancer in rats, *Cancer Lett.* 22 (1984) 99–102.
- [62] A. Subramanian, L. Kothari, Suppressive effect by melatonin on different phases of 9,10-dimethyl-1,2-benzanthracene (DMBA)-induced rat mammary gland carcinogenesis, *Anticancer Drugs* 2 (1991) 297–303.
- [63] A. Subramanian, L. Kothari, Melatonin, a suppressor of spontaneous murine mammary tumors, *J. Pineal Res.* 10 (1991) 136–140.
- [64] D.-X. Tan, B. Poeggeler, R.J. Reiter, L.D. Chen, S. Chen, L.C. Manchester, L.R. Barlow-Walden, The pineal hormone melatonin inhibits DNA-adduct formation induced by the chemical carcinogen safrole in vivo, *Cancer Lett.* 70 (1993) 65–71.
- [65] D.-X. Tan, R.J. Reiter, L.D. Chen, B. Poeggeler, L.C. Manchester, L.R. Barlow-Walden, Both physiological and pharmacological levels of melatonin reduce DNA adduct formation induced by the carcinogen safrole, *Carcinogenesis* 15 (1994) 215–218.
- [66] S.R. Teplitzky, T.L. Kiefer, Q. Cheng, P.D. Dwivedi, K. Moroz, L. Myers, M.B. Anderson, A. Collins, J. Dai, L. Yuan, L.L. Spriggs, D.E. Blask, S.M. Hill, Chemoprevention of NMU-induced rat mammary carcinoma with the combination of melatonin and 9-cis-retinoic acid, *Cancer Lett.* 26 (2001) 155–163.
- [67] K. Eck-Enriquez, T.L. Kiefer, L.L. Spriggs, S.M. Hill, Pathways through which a regimen of melatonin and retinoic acid induces apoptosis in MCF-7 human breast cancer cells, *Breast Cancer Res. Treat.* 61 (2000) 229–239.
- [68] M. Karbownik, R.J. Reiter, J. Cabrera, J.J. Garcia, Comparison of the protective effect of melatonin with other antioxidants in the hamster kidney model of estradiol-induced DNA damage, *Mutat. Res.* 474 (2001) 87–92.
- [69] R.T. Blickenstaff, S.M. Brandstadter, S. Reddy, R. Witt, Potential radioprotective agents. I. Homologues of melatonin, *J. Pharm. Sci.* 83 (1994) 216–218.
- [70] Vijayalaxmi, M.L. Meltz, R.J. Reiter, T.S. Herman, K.S. Kumar, Melatonin and protection from whole-body irradiation: survival studies in mice, *Mutat. Res.* 425 (1999) 21–27.
- [71] M. Karbownik, R.J. Reiter, W. Qi, J.J. Garcia, D.X. Tan, L.C. Manchester, Vijayalaxmi, Protective effects of melatonin against oxidation of guanine bases in DNA and decreased microsomal membrane fluidity in rat liver induced by whole body ionizing radiation, *Mol. Cell. Biochem.* 211 (2000) 137–144.
- [72] R.J. Reiter, Pineal melatonin: cell biology of its synthesis and of its physiological interactions, *Endocr. Rev.* 12 (1991) 151–158.
- [73] D.-X. Tan, L.C. Manchester, R.J. Reiter, W.B. Qi, M. Karbownik, J.R. Calvo, Significance of melatonin in antioxidant defense system: reactions and products, *Biol. Signals Recept.* 9 (2000) 137–159.
- [74] R. Hardeland, I. Blazer, B. Poeggeler, B. Fuhrberg, H. Uria, G. Behrmann, R. Wolf, T.J. Meyer, R.J. Reiter, On the primary functions of melatonin in evolution: Mediation of photoperiodic signals in a unicell, photooxidation and scavenging of free radicals, *J. Pineal Res.* 18 (1995) 104–111.
- [75] P. Stasica, P. Ulanski, J.M. Rosiak, Melatonin as a hydroxyl radical scavenger, *J. Radioanal. Nucl. Chem.* 232 (1998) 107–113.
- [76] B. Halliwell, J.M. Gutteridge, Oxygen free radicals and iron in relation to biology and medicine: some problems and concepts, *Arch. Biochem. Biophys.* 246 (1986) 501–514.
- [77] D.-X. Tan, L.C. Manchester, R.J. Reiter, B.F. Plummer, Cyclic 3-hydroxymelatonin: a melatonin metabolite generated as a result of hydroxyl radical scavenging, *Biol. Signals Recept.* 8 (1999) 70–74.
- [78] Y.H. Wu, W.G. Feenstra, J.N. Zhou, R.Y. Liu, J.S. Toranó, H.J. Van Kan, D.F. Fischer, R. Ravid, D.F. Swaab, Molecular changes underlying reduced pineal melatonin levels in Alzheimer disease: alterations in preclinical and clinical stages, *J. Clin. Endocrinol. Metab.* 88 (2003) 5898–5906.
- [79] W.E. Klunk, H. Engler, A. Nordberg, Y. Wang, G. Blomqvist, D.P. Holt, M. Bergstrom, I. Savitcheva, G.F. Huang, S. Estrada, B. Aussen, M.L. Debnath, J. Barletta, J.C. Price, J. Sandell, B.J. Lopresti, A. Wall, P. Koivisto, G. Antoni, C.A. Mathis, B. Langstrom, Imaging brain amyloid in Alzheimer's disease with Pittsburgh Compound-B, *Ann. Neurol.* 55 (2004) 306–319.
- [80] G.W. Small, V. Kepe, L.M. Ercoli, P. Siddarth, S.Y. Bookheimer, K.J. Miller, H. Lavretsky, A.C. Burggren, G.M. Cole, H.V. Vinters, P.M. Thompson, S.C. Huang, N. Satyamurthy, M.E. Phelps, J.R. Barrio, PET of brain amyloid and tau in mild cognitive impairment, *N. Engl. J. Med.* 355 (2006) 2652–2663.



# Reproductive and developmental effects of EMF in vertebrate animal models

Aris F. Pourlis\*

*Laboratory of Anatomy Histology & Embryology, Veterinary School, University of Thessaly Karditsa, Greece*

Received 10 August 2008; received in revised form 28 August 2008; accepted 30 January 2009

## Abstract

This paper reviews the literature data on the effects of electromagnetic fields (EMF), in the reproductive organs as well as in prenatal and postnatal development of vertebrate animals. Review articles which have been published till 2001, regarding the reproductive and developmental effects of the entire range of frequency of electromagnetic fields, were surveyed. Experimental studies which were published from 2001 onwards were summarized. Special focus on the effects of radiofrequencies related to mobile communication in the above mentioned topics has been made. According to the majority of the investigations, no strong effects resulted regarding the exposure to EMF of mobile telephony in the animal reproduction and development. However further research should be done in order to clarify many unknown aspects of the impact of EMF in the living organisms.

© 2009 Elsevier Ireland Ltd. All rights reserved.

**Keywords:** Electromagnetic fields (EMF); Mobile phones; Teratology; Endometrium; Testis

## 1. Introduction

During the 20th century, the exposure to electromagnetic fields (EMF) became an important source of concern about the possible effects in the living organisms. The artificial sources of electromagnetic radiation have risen tremendously because of the ongoing needs on electricity, telecommunications, and electronic devices. In this context, World Health Organisation (WHO) established in 1996 the International EMF project in order to assess health and environmental effects of exposure to EMF in the frequency range from 0 to 300 GHz. For the purpose of this paper this range will be divided into static (0 Hz), extremely low frequency (ELF > 0–300 kHz), intermediate frequencies (IF > 300–10 MHz) and radiofrequency (RF 10 MHz–300 GHz) fields [J. Juutilainen, Developmental effects of electromagnetic fields, *Bioelectromagnetics* 7 (2005) S107–S115]. The mobile phone technology is based on radiofrequency radiation with transmission of microwaves carrying frequencies between 880 and 1800 MHz [P.A. Valberg, T.E. van Deventer, M.H. Repacholi, Workgroup report:

base stations and wireless networks-radiofrequency (RF) exposures and health consequences, *Environ. Health Perspect.* 115 (2007) 416–424].

The mobile telephony revolution took place in the last decade. There is an increasing number of cell phone users all over the world. Also, new technologies which use the spectrum of high frequency emissions are incorporated in many aspects of telecommunications. As a consequence, there is a lot of interest about the possible effects of the radiation emitted from the machines which are engaged in the telephony such as hand phones, base stations and transmitters.

The biological effects of EMF have been and are being investigated on different levels of organization. On the level of human populations, epidemiological studies are used whereas, on the level of individuals human, animal and plant *in vivo* experiments are carried out. Furthermore, on the level of organs, tissues and cells *in vitro* investigations are employed. Finally, on the sub-cellular level, biochemical and molecular techniques are utilized.

From another point of view, many studies have been carried out or are in progress about the various effects of radiation emissions regarding the behaviour, cancer, central nervous system, sleep, children, cardiovascular system, immune function, reproduction and development [3].

\* Tel.: +30 24410 66013.

E-mail address: apourlis@vet.uth.gr.

The present paper will focus on the existing data about the reproductive and developmental effects of EMF in vertebrates. Reproduction is a critical function of the organisms and involves two body systems the male and female genital system. The development comprises a series of events which begins with fertilization, continues with implantation, embryonic growth and terms with sexual maturity. In the context of systematic zoology, the vertebrates are close to the humans. Therefore, the animal studies could provide useful information on the comprehension of interaction of EMF with the living organism and on the possible commonality with the humans.

The biological effects of EMF of interest can be broadly grouped into thermal and non-thermal [4]. The thermal effects are associated with local heat production just like the mechanism of a microwave oven. The non-thermal mechanism is triggered by an amount of energy absorption, which is not directly associated with temperature change but rather to some other changes produced in the tissues.

The goal of this paper is to present the up to date available data about the EMF and their potential effects on reproduction and development, filling the gap of information from the most recent published reviews. All the bibliographic data which will be presented were collected exclusively from scientific journals published in English and partially in other languages. The survey includes studies which were published from 2001 onward. The studies which relate to the impact of mobile phone electromagnetic fields will be presented thoroughly and independently from the date of their publication.

## 2. Historical background

The first paper which I found in the medical literature, regarding the effects of EMF on the development of vertebrates, was published in 1893 in an anatomical journal from Windle [5]. The author summarized the observations of three scientists and added his own about the effects of electricity on the chicken embryos. Two years later the same author [6], published an account on the effects of electricity and magnetism on development.

In 1980 two papers were published about the biological effects of microwave radiation. Cook et al. [7] published a comprehensive survey regarding the very early research on the biological effects of electromagnetic fields. The early work on short waves from 1885 to 1940 was presented. Following, the authors summarized the available data from 1940 to 1960. Leach [8] provided an account on the genetic, growth and reproductive effects of microwave radiation including early studies in this field that were published from 1959 to 1979. The majority of revised papers dealt with animals. Later, Algers and Hennichs [9] summarized the biological effects on vertebrates, of electromagnetic fields where the frequency did not exceed 100 Hz. The authors included many studies about the impact of EMF on farm animals. The same

year, a specialized review was published on the effects of non-ionizing radiation on birds [10].

Berman et al. [11], presented the results of a large multinational experimental effort (Henhouse project) regarding the low frequency EMF effects on chick embryos. Juutilainen [12], Chernoff et al. [13], Brent et al. [14] presented detailed reviews of the literature about the effects on reproduction related to low frequency EMF.

Jensh [15] reviewed behavioral teratologic studies using microwave radiation with special interest to continuous wave (CW) 915, 2450, or 6000 MHz radiation.

Verschaeve and Maes [16] reviewed the genetic, carcinogenic and teratogenic effects of RF (300 MHz–300 GHz). Regarding the effects on reproduction and teratogenesis, studies from 1961 to 1991 were surveyed. The majority of these experimental studies dealt with the exposure of animals at 2.45 GHz. The same year, Huuskonen et al. [17] reported on the teratogenic and reproductive effects of low frequency (0–100 kHz) magnetic fields associated with the use or transmission of electric power or emitted from video display terminals. The animal studies that were surveyed, have been published from 1987 to 1997 regarding the effects of alternating magnetic fields on prenatal development of rats and mice. In the same paper, studies on the effects of prenatal exposure of alternating magnetic fields on postnatal development were included. Brent [18] provided a thorough review of *in vivo* and *in vitro* studies on the reproductive and teratologic effects of low frequency EMF. The survey of reproductive effects has involved studies with chick embryos, chickens, cows, mice, and rats from 1969 to 1996. O'Connor [19] recorded the intrauterine effects in animals exposed to radiofrequency and microwave fields with a special feature. The SAR of the surveyed studies was above the limit of 0.4 W/kg.

Experimental studies on the teratologic effects or developmental abnormalities from exposure to RF electromagnetic fields in the range 3 kHz–300 GHz were reviewed from Heynick and Merritt [20]. The review included investigations with insects, birds (chicken, quails, turkeys) and mammalian species (mice, rats) as well as non-human primates which appeared from 1974 to 2000. A brief critical review on the developmental effects of extremely low frequency (ELF) electric and magnetic fields provided by Juutilainen [21]. Löscher [22] published a survey of the effects of radiofrequency electromagnetic fields on production, health and behaviour of farm animals.

Juutilainen [1] reported on the effects of EMF on animal development. In his review, he surveyed specific topics such as the Henhouse project, the interaction of LF-IMF EMF with known teratogens, and the behavioral teratology of RF. Saunders and McCaig [23] summarized the possible effects on prenatal development of physiologically weak electric fields induced in the body by exposure to extremely low frequency electromagnetic fields and of elevated temperature levels that might result from exposure to radiofrequency (RF) radiation.

Table 1  
Overview of investigations on EMF effects on animal genital system.

Animal species	Exposure frequency	Exposure parameters	Exposure duration	Endpoint	Results	Comments	Reference
Mouse Swiss	50 Hz	25 mT	Continuous 90 days	Effects on reproductive ability	No effect on the fertility of male and female mice. The ovarian weight was significantly increased		[27]
Mouse CD1 (BALB/c X DBA/2)	60 Hz	2 mT	Continuous for 72 h or 8 h/day for 10 days	Sperm morphology	No statistically differences were observed	Two groups were treated with mitomycin C. Sperm abnormalities were found in the group exposed versus the group treated with mitomycin C alone	[28]
Mouse BALB/c	60 Hz	0.1 or 0.5 mT	24 h/day for 8 weeks	Germ cell apoptosis in the testes	No significant changes in testicular weights. Decrease of normal seminiferous tubules. Increase of the germ cell death		[29]
Rat Sprague–Dawley	60 Hz	5, 83.3, 500 mT	Continuous 21 h/day from day 6 of gestation to day 21 of lactation	Spermatotoxicity and reproductive dysfunction in the F1 offspring	No detectable alterations in offspring spermatogenesis and fertility		[30]
Rat Sprague–Dawley	50 Hz	25 ± 1 µT	Continuous for 18 weeks	Effects on sperm count, weights of testes, seminal vesicles, preputial glands	No effect on the weight of testes. Significant reduction of the weight of seminal vesicles and preputial glands. Significant reduction in sperm count		[31]
Rat Sprague–Dawley	50 Hz	1.35 ± 0.018 mT	2 h/day, 7 days/week for 2 months	Sperm count, morphological changes of testes	No significant alterations were observed	Funding not mentioned	[32]
Rat Wistar albino ♂♂	50 Hz	1 mT (mean value)	3 h/day for 50 or 100 days	Morphological evaluation of uterus and ovaries	Ultrastructural alterations in germinal epithelium of ovaries in the experimental group (50 days) as well as in tunica albuginea (100 days)	Ambiguous observations in the uterus	[33]
Rat Sprague–Dawley ♂♀	20 kHz	6.25 mT	8 h/day, 5 days/week for 90 days	Histopathological examination of various organs	No differences were seen in testis and ovary		[34]

Table 1 (Continued)

Animal species	Exposure frequency	Exposure parameters	Exposure duration	Endpoint	Results	Comments	Reference
Rat Wistar ♂♀	50 Hz		3 weeks <i>in utero</i> and 5 weeks postnatal	Testes	Morphological changes in the boundary tissue of the seminiferous tubules		[35]
Rat Sprague–Dawley ♂♂	20 kHz sine waves	6.25 mT	8 h/day for 12 or 18 months	Histopathological examination of various organs	No differences were seen in testis and ovary		[36]
Rat Wistar	30–300 GHz	>0.3 mW/cm <sup>2</sup>	30 min for 63 days	Spermatogenesis	Morphological changes in spermatozoa	Scanty data presentation	[37]
Rat Wistar	50 Hz		8 h/day for 8 months	Histological evaluation of testes	Mean seminiferous tubule diameter and testicular weight were significantly lower in exposed group. Histologic damage score was threefold in experimental group versus control		[38]

A special topic, the effects of EMF from power lines on avian reproductive biology, was reviewed by Fernie and Reynolds [24]. Krewski et al. [25], reviewed studies referring to various disciplines regarding the effects of RF. The included literature was published between 2001 and 2003. A novelty of this paper, was a discussion of the reports of various authorities and committees about the potential health risks associated with exposure to RF fields. A gap in the literature regarding the biological effects of EMF in the intermediate frequency range was covered by the review of Shigemitsu et al. [26].

During the last decade, many reports from authorities (local, national and international) and expert panels have been uploaded on the web [2].

It is suggested that the reader refer to the above-mentioned review articles and electronic addresses, in order to assemble a more complete and detailed view of the biological effects of EMF.

### 3. Male genital system

The testes are very important organs situated externally to the body and enclosed by the scrotum. The testicular parenchyma is the site of an intense proliferation and differentiation of the germinal cells that will become the sperm cells. The testes are very sensitive to temperature variations and for this reason the scrotum, which contains the testicular parenchyma, has a specialized contractile structure.

Studies that have evaluated EMF effects (mainly LF) on the genital systems of the vertebrates are summarized in Table 1.

Regarding mobile telephony, the first study conducted by Dasdag et al. [39] investigated whether there are adverse effects due to microwave exposure emitted by cellular phones in male Wistar albino rats. The animals ( $n = 18$ ) were divided in three groups (control, standby exposed group, speech exposed group). Specific energy absorption rate (SAR) was 0.141 W/kg. Rats in the experimental groups were exposed for 2 h/day for 1 month in standby position, whereas phones were turned to the speech position three times for 1 min. The decrease of epididymal sperm counts in the speech groups was not found to be significant. Differences in terms of normal and abnormal sperm forms were not observed. Histological changes were especially observed in the testes of rats in the speech group. Seminiferous tubular diameter of rat testes in the standby and speech groups was found to be lower than the sham group. Rectal temperatures of rats in the speech group were found to be higher than the sham and standby groups. The rectal temperatures of rats before and after exposure were also found to be significantly higher in the speech group.

The same group of authors [40], failed to reproduce the results of their previous work. Sixteen Sprague–Dawley rats were separated into two groups (control, experimental). They were exposed to 890–915 MHz pulsed wave (PW) daily for



20 min/day for 1 month. For 250 mW average radiated power, SAR was 0.52 W/kg. No differences were observed in the percentages of epididymal normal and abnormal sperms, the epididymal sperm count, as well as in the seminiferous tubule diameter between control and experimental groups. Also, the testicular biopsy score as evaluated by Johnson's scale did not differ significantly.

Aitken et al. [41] assessed the testis of mice irradiated with 900 MHz in a waveguide, with an exposure condition SAR 90 mW/kg for 7 days at 12 h/day. The authors did not observe abnormalities regarding the sperm number, morphology and vitality. However, they reported significant damage to the mitochondrial genome as well as to the nuclear-globin locus.

Results similar to a previous study [39] regarding the diameter of the seminiferous tubules of rat testes were obtained by Ozguner et al. [42]. During the experiment, 20 male Sprague–Dawley rats (5 months of age) were either exposed to 900 MHz CW (average power density  $1 \pm 0.4$  mW/cm<sup>2</sup>) or not (control group). Rats exposed 30 min/day, for 5 days/week for 4 weeks. The authors also did not observe significantly different values of weight of testes, testicular biopsy score count and the percentage of interstitial tissue. However, the mean height of the germinal epithelium was found decreased in the group of rats that had been irradiated.

Forgács et al. [43] repeatedly exposed male NMRI mice to 1800 MHz GSM like microwave radiation at 0.018–0.023 W/kg whole body SAR. 11–12 sham exposed and 11–12 exposed mice were used. The animals were exposed ten times (over 2 weeks) and the duration of exposure was 2 h/day. No microwave exposure-related morphological alterations were found in testis, epididymis and prostate.

Adult male rats were examined after exposure at sub-chronic exposure to RF emitted from a conventional cell phone on their testicular function. Sixteen Wistar rats were used at age 30 days. The animals were exposed for 1 h daily during 11 weeks. The experimental group ( $n=8$ ) was exposed to 1835–1850 MHz at  $0.04\text{--}1.4$  mW/cm<sup>2</sup>. Total body weight and absolute and relative testicular and epididymal weights did not change significantly. Epididymal sperm count was not significantly different between the groups. Regarding the histomorphological endpoints of the study, no difference was found between the experimental and control arm [44].

The effect of cellular phone emissions on sperm characteristics in 16 Sprague–Dawley rats were studied [45]. The laboratory animals were divided in two groups (experimental, control) and exposed to four cell phones which had a personal communications service code division multiple access frequency band of 1.9 GHz (800 MHz digital and 800 MHz analog). The rats received daily (3 h–30 min rest–3 h) cell phone exposure for 18 weeks. The SAR ranged from 0.9 to 1.80 W/kg whereas the power from 0.00001 to 0.607 W, according to the specific mode of function. The authors analyzed the morphology of the sperm cells from

epididymis of rats. The percentage of deformities for the experimental group was 34.3% and the percentage of deformities for the control group was 32.1%. This difference in the occurrence of deformities between the two groups was not statistically significant ( $p>.05$ ) through a paired *t* test. The total sperm counts from the testes were not significantly different between the two groups. None of the temperature differences between the two groups were statistically significant.

Sixteen Sprague–Dawley rats were used to evaluate the bcl-2 protein (an anti-apoptotic protein) in rat testes. The experimental group ( $n=8$ ) was exposed to commercial (GSM) cellular phones irradiation for 20 min/day for 1 month. Average power density was measured at  $0.047$  mW/cm<sup>2</sup> and SAR levels changed between 0.29 and  $0.87$  W/kg. The testes were investigated by means of immunohistochemistry. No difference was observed between testes sections of the sham and experimental groups in terms of bcl-2 staining. These results indicate that the radiation emitted from 900 MHz cellular phones did not alter the anti-apoptotic protein in the testes of rats [46].

In order to investigate the apoptosis-inducing effect of mobile phone exposure on spermatogonia in seminiferous tubules, 31 Wistar albino male rats were divided in three groups such as cage control ( $n=10$ ), sham exposed ( $n=7$ ), and experimental ( $n=14$ ). The 2 h/day (7 days/week) exposure of 900 MHz radiation (power density  $0.012\text{--}0.149$  mW/cm<sup>2</sup> and SAR  $0.07\text{--}0.57$  W/kg) over a period of 10 months was evaluated by means of immunohistochemistry. The long-term radiation did not affect the active caspase-3 levels in testes of rats. Caspase-3 is a typical feature of apoptosis [47].

#### 4. Female genital system

Studies on the impact of RF in the female genital system are scarce. Two studies were conducted in order to evaluate the effects on endometrial apoptosis and the ameliorating effects of a combination of vitamin E and C against EMF damage.

Oral et al. [48], exposed sexually mature female rats (16 weeks old) to 900 MHz radiation, 30 min/day for 30 days. Twenty-four Wistar albino rats were divided in three groups (sham exposed, EMF exposed, EMF exposed treated with vitamin C and E). The animals were exposed at  $1.04$  mW/cm<sup>2</sup> (SAR  $0.016\text{--}4$  W/kg). The effect of microwaves was examined in rat endometrium by means of immunohistochemistry. Endometrial apoptosis was observed. Guney et al. [49], repeated the experiment with the addition of another group (control). Histological changes in endometrium, diffuse and severe apoptosis in the endometrial surface, epithelial and glandular cells were reported regarding the group exposed to EMF. Also, eosinophilic leucocyte and lymphocyte infiltration were seen in the endometrial stroma.

Table 2  
Overview of investigations on EMF effects on animal development.

Animal species	Exposure frequency	Exposure parameters	Exposure duration	Endpoint	Results	Comments	Reference
Rat Sprague–Dawley	50 Hz	7, 70, 350 mT	22 h/day during 0–7 or 8–15 day of gestation	Effects on teratogenicity and embryonic development	No differences regarding embryonic deaths, fetal weight and teratogenicity		[50]
Mouse ICR	50 Hz	Sham (0.1–1 $\mu$ T), 0.5, 5 mT	9 weeks $\sigma$ 2 weeks $\phi$ prior to mating	Effects on teratogenicity and embryonic development	No differences regarding embryonic deaths, fetal weight and teratogenicity		[51]
Mouse Swiss Webster	0 Hz–25 MHz		1 week beginning from the 18th day of pregnancy	Morphological changes in brain, thymus, adrenal gland during embryonic development	Pathological changes were observed in the neonates		[52]
Rat Sprague–Dawley	60 Hz	0 (sham group), 5, 83.3, 500 mT.	22 h/day during 6–20 day of gestation	Developmental toxicity	No differences regarding embryonic deaths, fetal weight and teratogenicity		[53]
Chicken	50 Hz	1.33–7.32 mT	24 h	Effects on teratogenicity and embryonic development	Significant difference in the percentage of abnormal embryos versus control was found in 4.19, 5.32, 5.86, and 6.65 densities. Some embryos with extra ribs, defects in ribs and vertebrae, anuria and abnormal beaks were observed	Funding not mentioned	[54]
Mouse ICR	20 kHz	6.5 mT	8 h/day from 2.5 to 15.5 days post-coitum	Effects on teratogenicity and embryonic development	No statistically significant differences in the number of implantation, embryonic death, resorption, growth retarded fetuses, external and skeletal abnormalities		[55]
Chicken Leghorn HR7	50 Hz	1 $\mu$ T, 500 $\mu$ T, 1 mT	Continuous for 15 or 21 days	Effects on embryo/fetus	At 15 days of incubation body weight was significantly lower versus controls. At 21 days of incubation the body weight and cranial diameters were smaller versus control. No differences in brain weight were observed in all groups	Funding not mentioned	[56]
Mouse $\phi$	Static magnetic field	400 mT	6 min/day from 7.5 to 14.5 day of pregnancy	Teratogenic effects	Polydactylism, abdominal fissure, fused ribs, vestigial 13th rib, brain hernia, curled tail		[57]
Mouse $\phi$	50 Hz	1.2 mT	8 h/day during pregnancy	Body weight of dams, development of offspring	Fetal loss, malformed fetuses, retardation of growth of the offspring in the first 2 weeks after birth	Article in chinese	[58]
Chicken White Leghorn eggs	50 Hz	1.33–7.32 mT	4 days	Morphological evaluation of embryos/fetuses	Abnormal brain cavities, spina bifida, monophthalmia, ocular defects and growth retardation		[59]

## 5. Developmental effects

The critical phases in the dynamic process of development take place mainly *in utero* (mammals) or *in ovo* (birds) i.e. during the embryonic period. The main bulk of investigations were performed regarding the possible effects on animals after irradiation, during *in utero* or *in ovo* development. The effects on development are determined by endpoints such as weight gain, congenital malformations, resorptions, and number of litters. These endpoints will be considered for various exposure conditions. The effects of EMF (mainly LF) on animal development are summarized in Table 2. Egg production was reduced (8%) when young laying hens have been continuously exposed to CW 915 MHz with an incident power of 800 mW during the first 2.5 weeks, 0 mW during the following week and 200 mW for the rest of experiment. Hatching of fertile and total eggs was not significantly influenced. No macroscopic malformations were observed in the chicks or dead embryos [60].

Jensh et al. [61] irradiated pregnant Wistar albino rats at a power density level of 10 mW/cm<sup>2</sup>, at a frequency of 915 MHz and average SAR 3.57 W/kg. The animals were exposed for 6 h/day from day 1 to day 21 of gestation. No significant teratogenic signs were observed regarding the resorption rate, malformation rate, mean litter size, fetal weight and number of live and dead fetuses. The experiment was repeated and extended in order to analyze the embryonic and postnatal development of offspring [62]. Eleven pregnant rats were irradiated and 19 rats were used as control animals. All animals delivered and raised their offspring (F<sub>1a</sub>) until weaning at 30 days of age. Ten days later females were rebred and teratologic evaluation was conducted on the resultant F<sub>1b</sub> fetuses. At 90 days of age, reproductive capability was evaluated and a standard teratologic analysis performed on the resultant F<sub>2</sub> offspring. No significant morphologic changes were revealed.

Pregnant rats were exposed at 970 MHz for 22 h/day from the 1st to 19th day of pregnancy [63]. The SAR values varied from 0.07, 2.4 and 4.8 W/kg. The embryo mortality, fetal weight, skeletal ossification, as well as maternal fertility were evaluated. The exposure with SAR 4.8 W/kg caused reduced (–12%) fetal body weight versus the control. All the other examined parameters were not significantly different.

Klug et al. [64] exposed rat embryos (9.5 days old) for up to 36 h to 900 MHz. The modulation frequency was fixed at 215 Hz and the SAR values were calculated at 0.2, 1 and 5 W/kg. The endpoints of the experiment were crown-rump length, number of somites as well as embryonic malformations. No significant changes were observed on the growth and differentiation parameters of the embryos. Magras and Xenos [65] investigated the possible effects of radiofrequency radiation on prenatal development in mice. The study consisted of *in vivo* experiments at several places around an “antenna park” where the frequency emissions ranged from 88.5 to 950 MHz. At these locations RF power densities between 168 and 1053 nW/cm<sup>2</sup> were measured. Twelve pairs

of mice, divided in two groups, were placed in locations of different power densities and were repeatedly mated five times. One hundred eighteen newborns were collected. They were measured, weighed, and examined macro- and microscopically. A progressive decrease in the number of newborns per dam was observed, which ended in irreversible infertility. The prenatal development of the newborns, however, evaluated by the crown-rump length, the body weight, and the number of the lumbar, sacral, and coccygeal vertebrae, was improved. Wistar albino rats [15] were exposed through pregnancy for 6 h each day to CW 915 MHz radiation at a power density level of 10 mW/cm<sup>2</sup>. Teratologic evaluation included the following parameters: mean litter size, maternal organ weight and organ weight/body weight ratios, body weight ratios of various organs (brain, liver, kidneys, and ovaries), number of resorptions and resorption rate, number of abnormalities and abnormality rate, mean term fetal weight. Mothers were rebred, and the second, unexposed litters were evaluated for teratogenic effects. Animals exposed to 915 MHz did not exhibit any consistent significant alterations in any of the above parameters.

Wistar rats were continuously exposed [66] during pregnancy to a low-level (0.1 mW/cm<sup>2</sup>) 900 MHz, 217 Hz pulse modulated EMF. Whole body average SAR values for the freely roaming, pregnant animals were measured in models; they ranged between 17.5 and 75 mW/kg. No differences between exposed and sham exposed dams or offspring were recorded in terms of litter size, evolution of body mass and developmental landmarks of litter mates. The effects of microwaves emitted by cellular phones on birth weights of rats were investigated by Dasdag et al. [67]. Thirty-six Wistar albino rats were divided into four groups. Each experimental or sham exposed group comprised six males or 12 females. The rats were exposed at 890–915 MHz (SAR 0.155 W/kg). Males were exposed daily for 3 × 1 min during 2 h/day for 1 month. Females were exposed in the same way until they gave birth. When the offspring became adult the experiment was repeated on them. No significant differences were observed between rectal temperatures in the sham and experimental groups. The birth weight of offspring in the experimental group was significantly lower than in the sham exposed group. However in the next generation of rats all parameters investigated were normal. Pregnant Sprague–Dawley rats were exposed [68] to ultra wide band (UWB) 0.1–1 GHz radiation in order to determine if teratological changes occur in rat pups as a result of (1) daily UWB exposures during gestation days 3 ± 18, or (2) as a result of both prenatal and postnatal (10 days) exposures. Dams were exposed either to (I) UWB irradiation with average whole body specific absorption rate 45 mW/kg (II) sham irradiation or (III) a positive control. Offspring were examined regarding litter size, sex-ratios, weights, coat appearance, and tooth eruption. The pups postnatally exposed were examined for hippocampal morphology. Generally, no significant differences were found between the exposed and sham group. The medial-to-lateral length of the hippocampus was significantly longer in the

Table 3

Summary of animal studies on effects of EMF (related to mobile telephony), on reproduction and development.

Animal species	Exposure frequency	Endpoint	Effect	Reference
Chicken	915 MHz	Development	No	[60]
Rat	915 MHz	Development	No	[61]
Rat	915 MHz	Development	No	[62]
Rat	970 MHz	Development	No	[63]
Rat	915 MHz	Development	No	[15]
Rat	900 MHz	Development	No	[64]
Mouse	88.5–950 MHz	Fertility/development	Yes/no	[65]
Rat	890–915 MHz	Testes	Yes	[39]
Rat	900 MHz	Development	No	[66]
Rat	0.1–1 GHz	Development	No	[68]
Rat	890–915 MHz	Development	Yes	[67]
Chicken	900 MHz	Development	Yes	[69]
Rat	890–915 MHz	Testes	No	[40]
Chicken		Development	Yes	[70]
Rat	900 MHz	Testes	No	[42]
Mouse	900 MHz	Testes	No	[41]
White stork	900–1800 MHz phone mast	Reproduction	Yes	[74]
Chicken	900 MHz	Kidney development	Yes	[71]
Mouse	1800 MHz	Testes	No	[43]
Rat	900 MHz	Endometrium	Yes	[48]
Rat	900 MHz	Brain development	No	[72]
Rat	1835–1850 MHz	Testes	No	[44]
Rat	1.9 GHz	Sperm	No	[45]
Tit	1200–3000 MHz	Reproduction	No	[75]
Rat	900 MHz	Endometrium	Yes	[49]
Chicken	900 MHz	Development	Yes	[73]
Rat	900 MHz	Testes	No	[46]
Rat	900 MHz	Testes	No	[47]

UWB-exposed pups than in the sham exposed animals but could not correlated with neurological dysfunction. The male offspring exposed *in utero* to UWB mated significantly less frequently than sham exposed males, but when they did mate there was no difference in fertilization and offspring numbers from the sham group.

Bastide et al. [69] reported chicken embryo mortality from day 7 to day 11 of incubation. This mortality reached 64% compared to 11% in controls. The maximum level of embryonic mortality was observed in the eggs placed near the telephone.

Chicken embryos were exposed to EMF from GSM mobile phone during the embryonic development [70]. The embryo mortality rate in the incubation period increased to 75% versus 16% in control group.

Ingole and Ghosh [71] studied by means of light microscopy the developmental effects on the avian kidney of radiation, from a cell phone handset (900 MHz frequency, power of 2 W and SAR of 0.37 W/kg). The authors reported morphological alterations on the epithelium of the renal tubules as well as of the renal corpuscles in E6, E8 and E10 chicken embryos.

The possible impact of cell phone radiation in the developing central nervous system of male Wistar rats was examined [72]. The animals were exposed to 900 MHz signal for 2 h/day on 5 days/week. After 5 weeks of exposure at whole body average SAR of 0.3 or 3 W/kg or sham exposure no degenerative morphological changes were found.

The results about the effects of exposing fertilized chicken eggs to a mobile phone over the entire period of incubation were published recently [73]. In this study, a series of 4 incubations were employed. During each incubation, 4 groups were used (control I, control II, experimental, sham). In the experimental group, the cell phone in call position was placed near ( $\leq 25$  cm) the eggs, whereas in the sham group the cell phone in off position was placed 1.5 m away from the exposed group. A significantly higher percentage of embryo mortality was observed in the experimental compared to the sham group in 2 of the 4 incubations. The lethal effects of embryo development in the experimental group were mainly observed between the 9th and 12th day of incubation.

Another issue that in recent years has attracted the attention of scientists is the effects of radiation from RF antennas on the biology of wild birds.

Balmori [74] investigated the possible effects of EMF from phone masts on a population of White stork (*Ciconia ciconia*). The total productivity in the nests located within 200 m of antennas was  $0.86 \pm 0.16$  versus  $1.6 \pm 0.14$  for those located further than 300 m. Another interesting observation, was that, 40% of the nests within 200 m of the antennae never had any chicks, while only 3.3% located further than 300 m never had chicks.

The influence of a military radar station [75] emitting pulsed modulated microwave radiation of 1200–3000 MHz was examined in tits (*Parus* sp). Experimental nest-boxes

were either exposed to a mean level of  $3.41 \pm 1.38$  or  $1.12 \pm 0.84 \text{ W/m}^2$ . For control nest-boxes the exposure ranged from 0.001 to  $0.01 \text{ W/m}^2$ . No statistically significant differences in the number of eggs or in the number of nestlings were observed between the two series (exposed, control) of tits.

## 6. Conclusions

The EMF were, are and will be a part of our life. The progress of science will provide the world with new EMF emitting technologies and subsequently with new problems. The monitoring of literature on this scientific field shows a shift of research which follows exactly the new technologies. The era of mobile telephony is beginning.

The evaluation of the possible effects of EMF on the living organism is a complex process that needs the combined contributions of many scientific disciplines. Due to the need for expertise in many different sciences, together with the technical problems of radiation studies, many times the published results are considered deficient in certain aspects. This is inevitable, and not an indication of poor quality. The inability to observe a biological effect in a particular study does not necessarily mean that such effect or/and adverse health effect is not present.

The vertebrate animal studies summarized in the present paper do not suggest strong effects of LF EMF on the male genital system. However, some studies on the development of animals, showed sensitivity, mainly observed in chickens. There is no convincing evidence from studies of mammals (Table 3), that exposure to EMF at levels associated with mobile telecommunications could be harmful for embryonic or postnatal development or for male fertility. On the other hand, the birds appeared to be more sensitive. The effects of EMF on the female genital system need further attention, since two experimental studies cannot lead to definitive conclusions.

The positive findings of the experimental studies with vertebrate animals are mainly attributed to the thermal effects of EMF. No valid evidence was found for the occurrence of non-thermal effects. However the non-thermal mechanisms must be the next target of the research.

The majority of reviewed studies were conducted in laboratories. This fact cannot represent the realistic situation of cell phone communication. On the other hand, the *in vivo* and simultaneously *in situ* studies are very scarce. Only Magras and Xenos conducted an *in situ* experiment which took place near an antenna park. That is because this kind of experiment is very difficult to carry out, and interaction with other exogenous factors could change the results.

One particular deficiency in most studies is that they describe experiments with acute or short-term exposure of animals on EMF. Experiments are needed to perform long-term exposure in order to demonstrate the chronic impact of EMF.

Another point that must be elucidated is that the majority of experimental animals used were small rodents (mice and rats), as well as chicken embryos. Further research is needed with the use of bigger animals such as dog and sheep.

The radiations emitted from masts that are situated in many rural and sylvatic areas could be possibly pathogenic in the wild animals. The wild animal populations could be candidate “experimental material” for closer observation of the possible effects of EMF on vertebrate models.

An important and intriguing aspect of the research is the possible role of the combination of RF with other pollutants such as chemical substances and other forms of radiation, as well as the interaction with drugs.

The potential health effects of EMF should be continually reassessed as new research results become available. EMF exposure guidelines also need to be updated or reconsidered as new scientific information on radiation and health risks is produced. However, additional studies might increase our understanding of the sensitivity of organisms to EMF.

## References

- [1] J. Juutilainen, Developmental effects of electromagnetic fields, *Bioelectromagnetics* 7 (2005) S107–S115.
- [2] P.A. Valberg, T.E. van Deventer, M.H. Repacholi, Workgroup report: base stations and wireless networks-radiofrequency (RF) exposures and health consequences, *Environ. Health Perspect.* 115 (2007) 416–424.
- [3] M.H. Repacholi, Health risks from the use of mobile phones, *Toxicol. Lett.* 120 (2001) 323–331.
- [4] L.J. Challis, Mechanisms for interaction between RF fields and biological tissue, *Bioelectromagnetics* 7 (2005) S98–S106.
- [5] B.C.A. Windle, On certain early malformations of the embryo, *J. Anat. Physiol.* 27 (1893) 436–453.
- [6] B.C.A. Windle, On the effects of electricity and magnetism on development, *J. Anat. Physiol.* 29 (1895) 346–351.
- [7] H.J. Cook, N.H. Steneck, A.J. Vander, G.L. Kane, Early research on the biological effects of microwave radiation: 1940–1960, *Ann. Sci.* 37 (1980) 323–351.
- [8] W.M. Leach, Genetic, growth and reproductive effects of microwave radiation, *Bull. N.Y. Acad. Med.* 56 (1980) 249–257.
- [9] B. Algers, K. Hennrichs, Biological effects of electromagnetic fields. A review, *Vet. Res. Commun.* 6 (1983) 265–279.
- [10] T.E. Bryan, R.P. Gildersleeve, Effects of nonionizing radiation on birds, *Comp. Biochem. Physiol.* 89A (1988) 511–530.
- [11] E. Berman, L. Chacon, D. House, Development of chicken embryos in a pulsed magnetic field, *Bioelectromagnetics* 11 (1990) 169–187.
- [12] J. Juutilainen, Effects of low-frequency magnetic fields on embryonic development and pregnancy, *Scand. J. Work Environ. Health.* 17 (1991) 149–158.
- [13] N. Chernoff, J.M. Rogers, R. Kavet, A review of the literature on potential reproductive and developmental toxicity of electric and magnetic fields, *Toxicology* 74 (1992) 91–126.
- [14] R.L. Brent, W.E. Gordon, W.R. Bennett, D.A. Beckman, Reproductive and teratologic effects of electromagnetic fields, *Reprod. Toxicol.* 7 (1993) 535–580.
- [15] R.P. Jensh, Behavioral teratological studies using microwave radiation: is there an increased risk from exposure to cellular phones and microwave ovens? *Reprod. Toxicol.* 11 (1997) 601–611.

- [16] L. Verschaeve, A. Maes, Genetic, carcinogenic and teratogenic effects of radiofrequency fields, *Mutation Res.* 410 (1998) 141–165.
- [17] H. Huuskonen, M.-L. Lindbohm, J. Juutilainen, Teratogenic and reproductive effects of low-frequency magnetic fields, *Mutation Res.* 410 (1998) 167–183.
- [18] R.L. Brent, Reproductive and teratologic effects of low-frequency electromagnetic fields: a review of *in vivo* and *in vitro* studies using animal models, *Teratology* 59 (1999) 261–286.
- [19] M.E. O'Connor, Mammalian teratogenesis and radiofrequency fields, *Proc. IEEE* 68 (1980) 56–60.
- [20] L.N. Heynick, J.H. Merritt, Radiofrequency fields and teratogenesis, *Bioelectromagnetics* 6 (2003) (2003) S174–S186.
- [21] J. Juutilainen, Developmental effects of extremely low frequency electric and magnetic fields, *Radiat. Prot. Dosim.* 106 (2003) 385–390.
- [22] W. Löscher, Die Auswirkungen elektromagnetischer Felder von Mobilfunkseanlagen auf Leistung, Gesundheit und Verhalten landwirtschaftlicher Nutztiere: Eine Bestandsaufnahme, *Der praktische Tierarzt* 84 (2003) 850–863.
- [23] R.D. Saunders, C.D. McCaig, Developmental effects of physiologically weak electric fields and heat: an overview, *Bioelectromagnetics* 7 (2005) S127–S132.
- [24] K.J. Fernie, S.J. Reynolds, The effects of electromagnetic fields from power lines on avian reproductive biology and physiology: a review, *J. Toxic Environ. Health B* 8 (2005) 127–140.
- [25] D. Krewski, B.W. Glickman, R.W.Y. Habash, B. Habbick, W.G. Lotz, R. Mandeville, F.S. Prato, T. Salem, D.F. Weaver, Recent advances in research on radiofrequency fields and health: 2001–2003, *J. Toxic Environ. Health B* 10 (2007) 287–318.
- [26] T. Shigemitsu, K. Yamazaki, S. Nakasono, M. Kakkikawa, A review of studies of the biological effects of electromagnetic fields in the intermediate frequency range, *IEEJ Trans. Electr. Electron. Eng.* 2 (2007) 405–412.
- [27] A. Elbetieha, M.A. Al-Akhras, H. Darmani, Long-term exposure of male and female mice to 50 Hz magnetic field: effects on fertility, *Bioelectromagnetics* 23 (2002) 168–172.
- [28] J.A. Heredia-Rojas, D.E. Caballero-Hernandez, A.O. Rodriguez-De La Fuente, G. Ramos-Alfano, L.E. Rodriguez-Flores, Lack of alterations on meiotic chromosomes and morphological characteristics of male germ cells in mice exposed to a 60 Hz and 2.0 mT magnetic field, *Bioelectromagnetics* 25 (2004) 63–68.
- [29] J.S. Lee, S.S. Ahn, K.C. Jung, Y.W. Kim, S.K. Lee, Effects of 60 Hz electromagnetic field exposure on testicular germ cell apoptosis in mice, *Asian J. Androl.* 6 (2004) 29–34.
- [30] M.K. Chung, S.J. Lee, Y.B. Kim, S.C. Park, D.H. Shin, S.H. Kim, J.C. Kim, Evaluation of spermatogenesis and fertility in F1 male rats after *in utero* and neonatal exposure to extremely low frequency electromagnetic fields, *Asian J. Androl.* 7 (2005) 189–194.
- [31] M.A. Al-Akhras, H. Darmani, A. Elbetieha, Influence of 50 Hz magnetic field on sex hormones and other fertility parameters of adult male rats, *Bioelectromagnetics* 27 (2006) 127–131.
- [32] M.Z. Akdag, S. Dasdag, F. Aksen, B. Isik, F. Yilmaz, Effect of ELF magnetic fields on lipid peroxidation, sperm count, p53 and trace elements, *Med. Sci. Monit.* 12 (2006) 366–371.
- [33] F. Aksen, M.Z. Akdag, A. Ketani, B. Yokus, A. Kaya, S. Dasdag, Effect of 50 Hz 1 mT magnetic field on the uterus and ovaries of rats, *Med. Sci. Monit.* 12 (2006) 215–220.
- [34] S.H. Kim, H.J. Lee, S.Y. Choi, Y.M. Gimm, J.K. Pack, H.D. Choi, Y.S. Lee, Toxicity bioassay in Sprague–Dawley rats exposed to 20 kHz triangular magnetic field for 90 days, *Bioelectromagnetics* 27 (2006) 105–111.
- [35] A.A. Khaki, R.S. Tubbs, M.M. Shoja, J.S. Rad, A. Khaki, R.M. Farahani, S. Zarrintan, T.C. Nag, The effects of an electromagnetic field on the boundary tissue of the seminiferous tubules of the rat: a light and transmission electron microscope study, *Folia Morphol.* 65 (2006) 188–194.
- [36] H.J. Lee, S.H. Kim, S.Y. Choi, Y.M. Gimm, J.K. Pack, H.D. Choi, Y.S. Lee, Long-term exposure of Sprague–Dawley rats to 20 kHz triangular magnetic fields, *Int. J. Radiat. Biol.* 82 (2006) 285–291.
- [37] T.I. Subbotina, O.V. Tereshkina, A.A. Khadartsev, A.A. Yashin, Effect of low-intensity extremely high frequency radiation on reproductive function in Wistar rats, *Bull. Exp. Biol. Med.* 142 (2006) 189–190.
- [38] S. Erpek, M.D. Bilgin, E. Dikicioglu, A. Karul, The effects of low frequency electric field in rat testis, *Revue Méd. Vét* 158 (2007) 206–212.
- [39] S. Dasdag, M.A. Ketani, Z. Akdag, A.R. Ersay, I. Sari, O.C. Demirtas, M.S. Celik, Whole body microwave exposure emitted by cellular phones and testicular function of rats, *Urol. Res.* 27 (1999) 219–223.
- [40] S. Dasdag, M.Z. Akdag, F. Aksen, F. Yilmaz, M. Bashan, M.M. Dasdag, M.S. Celik, Whole body exposure of rats to microwaves emitted from a cell phone does not affect the testes, *Bioelectromagnetics* 24 (2003) 182–188.
- [41] R.J. Aitken, L.E. Bennetts, D. Sawyer, A.M. Wiklundt, B.V. King, Impact of radio frequency electromagnetic radiation on DNA integrity in the male germ line Inter, *J. Androl.* 28 (2005) 171–179.
- [42] M. Ozguner, A. Koyu, G. Cesur, M. Ural, F. Ozguner, A. Gokcimen, N. Delibas, Biological and morphological effects on the reproductive organ of rats after exposure to electromagnetic field, *Saudi Med. J.* 26 (2005) 405–410.
- [43] Z. Forgács, Z. Somosy, G. Kubinyi, J. Bakos, A. Hudák, A. Surján, G. Thuróczy, Effects of whole body 1800 MHz GSM-like microwave exposure on testicular steroidogenesis and histology in mice, *Reprod. Toxicol.* 22 (2006) 111–117.
- [44] E.P. Ribeiro, E.L. Rhoden, M.M. Horn, C. Rhoden, L.P. Lima, L. Toniolo, Effects of sub chronic exposure to radio frequency from a conventional cellular telephone on testicular function in adult rats, *J. Urol.* 177 (2007) 395–399.
- [45] J.G. Yan, M. Agresti, T. Bruce, Y.H. Yan, A. Granlund, H.S. Matloub, Effects of cellular phone emissions on sperm motility in rats, *Fertil. Steril.* 88 (2007) 957–964.
- [46] F. Yilmaz, S. Dasdag, M.Z. Akdag, N. Kilinc, Whole body exposure of radiation emitted from 900 MHz mobile phones does not seem to affect the levels of anti-apoptotic bcl-2 protein, *Electromagn. Biol. Med.* 27 (2008) 65–72.
- [47] S. Dasdag, M.Z. Akdag, E. Ulukaya, A.K. Uzunlar, D. Yegin, Mobile phone exposure does not induce apoptosis on spermatogenesis in rats, *Arch. Med. Res.* 39 (2008) 40–44.
- [48] B. Oral, M. Guney, F. Ozguner, N. Karahan, T. Mungan, S. Comlekci, G. Cesur, Endometrial apoptosis induced by a 900 MHz mobile phone: preventive effects of vitamins E and C, *Adv. Ther.* 23 (2006) 957–973.
- [49] M. Guney, F. Ozguner, B. Oral, N. Karahan, T. Mungan, 900 MHz radiofrequency induced histopathologic changes and oxidative stress in rat endometrium: protection by vitamins E and C, *Toxicol. Ind. Health* 23 (2007) 411–420.
- [50] T. Negishi, S. Imai, M. Itabashi, I. Nishimura, T. Sasano, Studies of 50 Hz circularly polarized magnetic fields of up to 350 mT on reproduction and embryo-fetal development in rats: exposure during organogenesis or during preimplantation, *Bioelectromagnetics* 23 (2002) 369–389.
- [51] Y. Ohnishi, F. Mizuno, T. Sato, M. Yasui, T. Kikuchi, M. Ogawa, Effects of power frequency alternating magnetic fields on reproduction and prenatal development of mice, *J. Toxicol. Sci.* 27 (2002) 131–138.
- [52] Z.E. Gagnon, C. Newkirk, J.A. Conetta, M.A. Sama, S. Sisselman, Teratogenic effect of broad-band electromagnetic field on neonatal mice, *J. Environ. Sci. Health A Tox. Hazard Subst. Environ. Eng.* 38 (2003) 2465–2481.
- [53] M.K. Chung, J.C. Kim, S.H. Myung, D.I. Lee, Developmental toxicity evaluation of ELF magnetic fields in Sprague–Dawley rats, *Bioelectromagnetics* 24 (2003) 231–240.
- [54] M.S. Lahijani, K. Sajadi, Development of preincubated chicken eggs following exposure to 50 Hz electromagnetic fields with 1.33–7.32 mT flux densities, *Indian J. Exp. Biol.* 42 (2004) 858–865.
- [55] S.H. Kim, J.E. Song, S.R. Kim, H. Oh, Y.M. Gimm, D.S. Yoo, J.K. Pack, Y.S. Lee, Teratological studies of prenatal exposure of mice to a



- 20 kHz sawtooth magnetic field, *Bioelectromagnetics* 25 (2004) 114–117.
- [56] O. Roda-Murillo, J.A. Roda-Moreno, M.T. Morente-Chiquero, Effects of low-frequency magnetic fields on different parameters of embryo of *Gallus domesticus*, *Electromagn. Biol. Med.* 24 (2005) 55–62.
- [57] K. Saito, H. Suzuki, K. Suzuki, Teratogenic effects of static magnetic field on mouse fetuses, *Reprod. Toxicol.* 22 (2006) 118–124.
- [58] Y.N. Cao, Y. Zhang, Y. Liu, Effects of exposure to extremely low frequency electromagnetic fields on reproduction of female mice and development of offspring, *Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi* 24 (2006) 468–470.
- [59] M.S. Lahijani, S.E. Nojooshi, S.F. Siadat, Light and electron microscope studies of effects of 50 Hz electromagnetic fields on preincubated chick embryo, *Electromagn. Biol. Med.* 26 (2007) 83–98.
- [60] W.F. Krueger, A.J. Giarola, J.W. Bradley, A. Shrekenhamer, Effects of electromagnetic fields on fecundity in the chicken, *Ann. N.Y. Acad. Sci.* 247 (1975) 391–400.
- [61] R.P. Jensh, I. Weinberg, R.I. Brent, Teratologic studies of prenatal exposure of rats to 915 MHz microwave radiation, *Radiat. Res.* 92 (1982) 160–171.
- [62] R.P. Jensh, W.H. Vogel, R.I. Brent, Postnatal functional analysis of prenatal exposure of rats to 915 MHz microwave radiation, *Int. J. Toxicol.* 1 (1982) 73–90.
- [63] E. Berman, C. Weil, P.A. Philips, H.B. Carter, D.E. House, Fetal and maternal effects of continual exposure of rats to 970 MHz circularly-polarized microwaves, *Electromagn. Biol. Med.* 11 (1992) 43–54.
- [64] S. Klug, M. Hetscher, S. Giles, S. Kohlsman, K. Kramer, The lack of effects of nonthermal RF electromagnetic fields on the development of rat embryos grown in culture, *Life Sci.* 61 (1997) 1789–1802.
- [65] I.N. Magras, T.D. Xenos, RF radiation-induced changes in the prenatal development of mice, *Bioelectromagnetics* 18 (1997) 455–461.
- [66] M. Bornhausen, H. Scheingraber, Prenatal exposure to 900 MHz cell phone electromagnetic fields had no effect on operant-behaviour performances of adult rats, *Bioelectromagnetics* 21 (2000) 566–574.
- [67] S. Dasdag, M.Z. Akdag, O. Ayyildiz, O.C. Demirtas, M. Yayla, C. Sert, Do cellular phones alter blood parameters and birth weight of rats? *Electromagn. Biol. Med.* 19 (2000) 107–113.
- [68] B.L. Cobb, J.R. Jauchem, P.A. Mason, M.P. Dooley, S.A. Miller, J.M. Ziriak, M.R. Murphy, Neural and behavioural teratological evaluation of rats exposed to Ultra-Wideband electromagnetic fields, *Bioelectromagnetics* 21 (2000) 524–537.
- [69] M. Bastide, B.J. Youbicier-Simoes, J.C. Lebecq, J. Giaimis, Toxicologic study of electromagnetic radiation emitted by television and video display screens and cellular telephones on chickens and mice, *Indoor Built Environ.* 10 (2001) 291–298.
- [70] Y. Grigoryev, Biological effects of mobile phone electromagnetic field on chick embryo (Risk assessment using the mortality rate), *Radiats. Biol. Radioecol.* 43 (2003) 541–543.
- [71] I.V. Ingole, S.K. Ghosh, Cell phone radiation and developing tissues in chicken embryo—a light microscopic study of kidneys, *J. Anat. Soc. India* 55 (2006) 19–23.
- [72] T. Kumlin, H. Iivonen, P. Miettinen, A. Junonen, T. van Groen, L. Puranen, R. Pitkäaho, R. Juutilainen, H. Tanila, Mobile phone radiation and the developing brain: behavioral and morphological effects in juvenile rats, *Radiat. Res.* 168 (2007) 471–479.
- [73] F. Batellier, I. Couty, D. Picard, J.P. Brillard, Effects of exposing chicken eggs to a cell phone in ‘call’ position over the entire incubation period, *Theriogenology* 69 (2008) 737–745.
- [74] A. Balmori, Possible effects of electromagnetic fields from phone masts on a population of White stork (*Ciconia ciconia*), *Electromagn. Biol. Med.* 24 (2005) 109–119.
- [75] L. Rejt, T. Mazgajski, R. Kubacki, J. Kieliszek, E. Sobiczewska, S. Szmigielski, Influence of radar radiation on breeding biology of tits (*Parus* sp), *Electromagn. Biol. Med.* 26 (2007) 235–238.



ELSEVIER

Pathophysiology xxx (2009) xxx–xxx

ISP  
PATHOPHYSIOLOGY

www.elsevier.com/locate/pathophys

# Electromagnetic pollution from phone masts. Effects on wildlife

Alfonso Balmori

*Dirección General del Medio Natural, Consejería de Medio Ambiente, Junta de Castilla y León, C/Rigoberto Cortejoso,  
14, 47014 Valladolid, Spain*

Received 10 August 2008; received in revised form 28 August 2008; accepted 30 January 2009

## Abstract

A review on the impact of radiofrequency radiation from wireless telecommunications on wildlife is presented. Electromagnetic radiation is a form of environmental pollution which may hurt wildlife. Phone masts located in their living areas are irradiating continuously some species that could suffer long-term effects, like reduction of their natural defenses, deterioration of their health, problems in reproduction and reduction of their useful territory through habitat deterioration. Electromagnetic radiation can exert an aversive behavioral response in rats, bats and birds such as sparrows. Therefore microwave and radiofrequency pollution constitutes a potential cause for the decline of animal populations and deterioration of health of plants living near phone masts. To measure these effects urgent specific studies are necessary.

© 2009 Published by Elsevier Ireland Ltd.

**Keywords:** Effects on wildlife; Effects on birds; Electromagnetic radiation; Mammals; Microwaves; Mobile telecommunications; Non-thermal effects; Phone masts; Radiofrequencies

## 1. Introduction

Life has evolved under the influence of two omnipresent forces: gravity and electromagnetism. It should be expected that both play important roles in the functional activities of organisms [1]. Before the 1990's radiofrequencies were mainly from a few radio and television transmitters, located in remote areas and/or very high places. Since the introduction of wireless telecommunication in the 1990's the rollout of phone networks has caused a massive increase in electromagnetic pollution in cities and the countryside [2,3].

Multiple sources of mobile communication result in chronic exposure of a significant part of the wildlife (and man) to microwaves at non-thermal levels [4]. In recent years, wildlife has been chronically exposed to microwaves and RFR (Radiofrequency radiation) signals from various sources, including GSM and UMTS/3G wireless phones and base stations, WLAN (Wireless Local Area Networks), WPAN (Wireless Personal Area Networks such as Bluetooth), and DECT (Digital Enhanced (former European) Cordless Telecommunications) that are erected indiscriminately without studies of environmental impact measuring

long-term effects. These exposures are characterized by low intensities, varieties of signals, and long-term durations. The greater portion of this exposure is from mobile telecommunications (geometric mean in Vienna: 73% [5]). In Germany the GSM cellular phone tower radiation is the dominating high frequency source in residential areas [6]. Also GSM is the dominating high frequency source in the wilderness of Spain (personal observation).

Numerous experimental data have provided strong evidence of athermal microwave effects and have also indicated several regularities in these effects: dependence of frequency within specific frequency windows of "resonance-type"; dependence on modulation and polarization; dependence on intensity within specific intensity windows, including super-low power density comparable with intensities from base stations/masts [4,7–9]. Some studies have demonstrated different microwave effects depending on wavelength in the range of mm, cm or m [10,11]. Duration of exposure may be as important as power density. Biological effects resulting from electromagnetic field radiation might depend on dose, which indicates long-term accumulative effects [3,9,12]. Modulated and pulsed radiofrequencies seem to be more effective in producing effects [4,9]. Pulsed waves (in blasts), as well as certain low frequency modulations exert greater

E-mail addresses: abalmori@ono.com, balmoral@jcy.es.

0928-4680/\$ – see front matter © 2009 Published by Elsevier Ireland Ltd.  
doi:10.1016/j.pathophys.2009.01.007

Please cite this article in press as: A. Balmori, Electromagnetic pollution from phone masts. Effects on wildlife, Pathophysiology (2009), doi:10.1016/j.pathophys.2009.01.007

biological activity [11,13–15]. This observation is important because cell phone radiation is pulsed microwave radiation modulated at low frequencies [8,9].

Most of the attention on possible biological effects of electromagnetic radiation from phone masts has been focused on human health [5,16–21]. The effects of electromagnetic pollution on wildlife, have scarcely been studied [22–25].

The objective of this review is to detail advances in knowledge of radiofrequencies and microwave effects on wildlife. Future research may help provide a better understanding of electromagnetic field (EMF) effects on wildlife and plants and their conservation.

## 2. Effects on exposed wildlife

### 2.1. Effects on birds

#### 2.1.1. Effects of phone mast microwaves on white stork

In monitoring a white stork (*Ciconia ciconia*) population in Valladolid (Spain) in vicinity of Cellular Phone Base Stations, the total productivity in nests located within 200 m of antennae, was  $0.86 \pm 0.16$ . For those located further than 300 m, the result was practically doubled, with an average of  $1.6 \pm 0.14$ . Very significant differences among total productivity were found ( $U = 240$ ;  $P = 0.001$ , Mann–Whitney test). Twelve nests (40%) located within 200 m of antennae never had chicks, while only one (3.3%) located further than 300 m had no chicks. The electric field intensity was higher on nests within 200 m ( $2.36 \pm 0.82$  V/m) than nests further than 300 m ( $0.53 \pm 0.82$  V/m). In nesting sites located within 100 m of one or several cellsite antennae with the main beam of radiation impacting directly (Electric field intensity  $> 2$  V/m) many young died from unknown causes. Couples frequently fought over nest construction sticks and failed to advance the construction of the nests. Some nests were never completed and the storks remained passively in front of cellsite antennae. These results indicate the possibility that microwaves are interfering with the reproduction of white stork [23]. (Fig. 1)

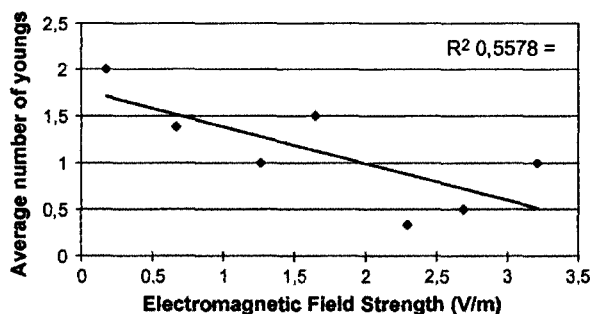


Fig. 1. Average number of young and electric field intensity (V/m) in 60 nests of white storks (*Ciconia ciconia*) (Hallberg, Ö with data of Balmori, 2005 [23]).

#### 2.1.2. Effects of phone mast microwaves on house sparrows

A possible effect of long-term exposure to low-intensity electromagnetic radiation from mobile phone (GSM) base stations on the number of house sparrows during the breeding season was studied in Belgium. The study was carried out sampling 150 point locations within six areas to examine small-scale geographic variation in the number of house sparrow males and the strength of electromagnetic radiation from base stations. Spatial variation in the number of house sparrow males was negative and highly significantly related to the strength of electric fields from both the 900 and 1800 MHz downlink frequency bands and from the sum of these bands (Chi-square-tests and AIC-criteria,  $P < 0.001$ ). This negative relationship was highly similar within each of the six study areas, despite differences among areas in both the number of birds and radiation levels. Fewer house sparrow males were seen at locations with relatively high electric field strength values of GSM base stations and therefore support the notion that long-term exposure to higher levels of radiation negatively affects the abundance or behavior of house sparrows in the wild [24].

In another study with point transect sampling performed at 30 points visited 40 times in Valladolid (Spain) between 2002 and 2006, counting the sparrows and measuring the mean electric field strength (radiofrequencies and microwaves: 1 MHz to 3 GHz range). Significant declines ( $P = 0.0037$ ) were observed in mean bird density over time, and significantly low bird density was observed in areas with high electric field strength. The logarithmic regression of the mean bird density vs. field strength groups (considering field strength in 0.1 V/m increments) was  $R = -0.87$ ;  $P = 0.0001$ . According to this calculation, no sparrows would be expected to be found in an area with field strength  $> 4$  V/m [25]. (Fig. 2)

In the United Kingdom a decline of several species of urban birds, especially sparrows, has recently happened [26]. The sparrow population in England has decreased in the last 30 years from 24 million to less than 14. The more abrupt decline, with 75% descent has taken place from 1994 to 2002. In 2002, the house sparrow was added to the Red List of U.K. endangered species [27]. This coincides with the rollout of mobile telephony and the

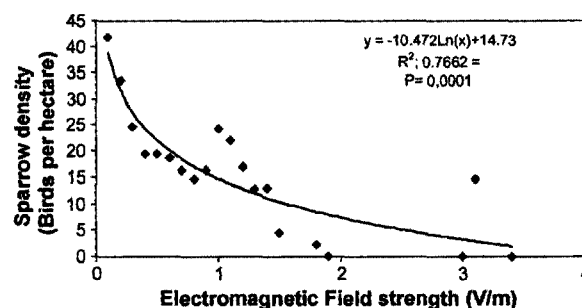


Fig. 2. Mean sparrow density as a function of electric field strength grouped in 0.1 V/m. (Balmori and Hallberg, 2007 [25]).

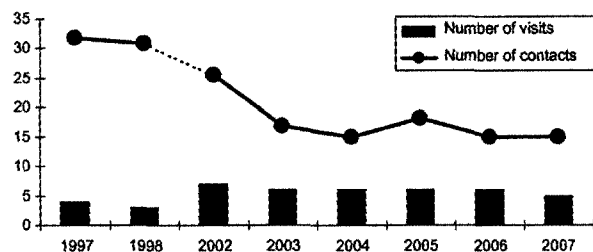


Fig. 3. Annual number of contacts (Mean) for 14 species studied in “Campo Grande” urban park (lack the information of the years 1999–2001).

possible relationship of both circumstances should be investigated.

In Brussels, many sparrows have disappeared recently [28]; similar declines have been reported in Dublin [29]. Van der Poel (cited in Ref. [27]) suggested that sparrows might be declining in Dutch urban centres also.

#### 2.1.3. Effects on the bird community at an urban park

Microwaves may be affecting bird populations in places with high electromagnetic pollution. Since several antennas were installed in proximities of “Campo Grande” urban park (Valladolid, Spain) the bird population has decreased and a reduction of the species and breeding couples has occurred. Between 1997 and 2007, of 14 species, 3 species have disappeared, 4 are in decline and 7 stay stable (Balmori, unpublished data) (Fig. 3). In this time the air pollution (SO<sub>2</sub>, NO<sub>2</sub>, CO and Benzene) has diminished.

During the research some areas called “silence areas” contaminated with high microwave radiation (>2 V/m), where previously different couples usually bred and later disappeared, have been found. Several anomalies in magpies (*Pica pica*) were detected: plumage deterioration, locomotive problems (limps and deformations in the paws), partial albinism and melanism, especially in flanks [30]. Recently cities have increased cases of partial albinism and melanism in birds (*Passer domesticus*, *Turdus merula* and *P. pica*) (personal observation).

#### 2.1.4. Possible physiological mechanisms of the effects found in birds

Current scientific evidence indicates that prolonged exposure to EMFs, at levels that can be encountered in the environment, may affect immune system function by affecting biological processes [3,31,32]. A stressed immune system may increase the susceptibility of a bird to infectious diseases, bacteria, viruses, and parasites [33].

The plumage of the birds exposed to microwaves looked, in general, discolored and lack of shine. This not only occurred in ornamental birds; such as peacocks, but also in wild birds; such as, tits, great tits, house sparrows, etc (personal observation). We must mention that plumage deterioration is the first sign of weakening or illnesses in birds since damaged feathers are a sure sign of stress.

Physiological conditions during exposure minimize microwave effects. Radical scavengers/antioxidants might be involved in effects of microwaves [4].

Microwaves used in cellphones produce an athermal response in several types of neurons of the birds nervous system [34]. Several studies addressed behavior and teratology in young birds exposed to electromagnetic fields [23,25,35–37]. Most studies indicate that electromagnetic field exposure of birds generally changes, but not always consistently in effect or in direction, their behavior, reproductive success, growth and development, physiology and endocrinology, and oxidative stress [37]. These results can be explained by electromagnetic fields affecting the birds’ response to the photoperiod as indicated by altered melatonin levels [38].

Prolonged mobile phone exposure may have negative effects on sperm motility characteristics and male fertility as has been demonstrated in many studies made in man and rats [39–46]. EMF and microwaves can affect reproductive success in birds [23,25,35,36,47]. EMF exposure affected reproductive success of kestrels (*Falco sparverius*), increasing fertility, egg size, embryonic development and fledging success but reducing hatching success [35,36].

The radiofrequency and microwaves from mobile telephony can cause genotoxic effects [48–55]. Increases in cytological abnormalities imply long-term detrimental effects since chromosomal damage is a mechanism relevant to causation of birth defects and cancer [55].

Long-term continuous, or daily repeated EMF exposure can induce cellular stress responses at non-thermal power levels that lead to an accumulation of DNA errors and to inhibition of cell apoptosis and cause increased permeability of blood–brain barrier due to stabilization of endothelial cell stress fibers. Repeated occurrence of these events over a long period of time (years) could become a health hazard due to a possible accumulation of brain tissue damage. These findings have important implications with regards to potential dangers from prolonged and repeated exposure to non-ionizing radiation [56,57].

Pulsed magnetic fields can have a significant influence on the development and incidence of abnormalities in chicken embryos. In five of six laboratories, exposed embryos exhibited more structural anomalies than controls. If the data from all six laboratories are pooled, the difference for the incidence of abnormalities in exposed embryos and controls is highly significant [58]. Malformations in the nervous system and heart, and delayed embryo growth are observed. The embryo is most sensitive to exposure in the first 24 h of incubation [58]. An increase in the mortality [59] and appearance of morphological abnormalities, especially of the neural tube [13,60,61] has been recorded in chicken embryos exposed to pulsed magnetic fields, with different susceptibility among individuals probably for genetic reasons. A statistically significant high mortality rate of chicken embryos subjected to radiation from a cellphone, compared to the control group exists [62,63]. In another study eggs exposed to a magnetic

field intensity of 0.07 T showed embryonic mortality during their incubation was higher. The negative effect of the magnetic field was manifested also by a lower weight of the hatched chicken [64]. Bioelectric fields have long been suspected to play a causal role in embryonic development. Alteration of the electrical field may disrupt the chemical gradient and signals received by embryo cells. It appears that in some manner, cells sense their position in an electrical field and respond appropriately. The disruption of this field alters their response. Endogenous current patterns are often correlated with specific morphogenetic events [65].

Available data suggests dependencies of genotype, gender, physiological and individual factors on athermal microwave effects [4,9]. Genomic differences can influence cellular responses to GSM Microwaves. Data analysis has highlighted a wide inter-individual variability in response, which was replicated in further experiments [4]. It is possible that each species and each individual, show different susceptibility to radiation, since vulnerability depends on genetic tendency, and physiologic and neurological state of the irradiated organism [15,35–37,61,66–68]. Different susceptibility of each species has also been proven in wild birds exposed to electromagnetic fields from high-voltage power lines [47].

## 2.2. Effects on mammals

### 2.2.1. Alarm and aversion behavior

Rats spent more time in the halves of shuttle boxes that were shielded from 1.2 GHz. Microwaves irradiation. The average power density was about 0.6 mW/cm<sup>2</sup>. Data revealed that rats avoided the pulsed energy, but not the continuous energy, and less than 0.4 mW/cm<sup>2</sup> average power density was needed to produce aversion [69]. Navakatikian & Tomashevskaya [70] described a complex series of experiments in which they observed disruption of rat behavior (active avoidance) from radiofrequency radiation. Behavioral disruption was observed at a power density as low as 0.1 mW/cm<sup>2</sup> (0.027 W/kg). Mice in an experimental group exposed to microwave radiation expressed visible individual panic reaction, disorientation and a greater degree of anxiety. In the sham exposed group these deviations of behavior were not seen and all animals show collective defense reaction [71]. Microwave radiation at 1.5 GHz pulsing 16 ms. At 0.3 mW/cm<sup>2</sup> power density, in sessions of 30 min/day over one month produced anxiety and alarm in rabbits [72].

Electromagnetic radiation can exert an aversive behavioral response in bats. Bat activity is significantly reduced in habitats exposed to an electromagnetic field strength greater than 2 V/m [73]. During a study in a free-tailed bat colony (*Tadarida teniotis*) the number of bats decreased when several phone masts were placed 80 m from the colony [74].

### 2.2.2. Deterioration of health

Animals exposed to electromagnetic fields can suffer a deterioration of health and changes in behavior [75,76].

There was proof of frequent death in domestic animals; such as, hamsters and guinea pigs, living near mobile telecommunication base stations (personal observation).

The mice in an experimental group exposed to microwave radiation showed less weight gain compared to control, after two months. The amount of food used was similar in both groups [71]. A link between electromagnetic field exposure and higher levels of oxidative stress appears to be a major contributor to aging, neurodegenerative diseases, immune system disorders, and cancer in mammals [33].

The effects from GSM base transceiver station (BTS) frequency of 945 MHz on oxidative stress in rats were investigated. When EMF at a power density of 3.67 W/m<sup>2</sup>, below current exposure limits, were applied, MDA (malondialdehyde) level was found to increase and GSH (reduced glutathione) concentration was found to decrease significantly ( $P < 0.0001$ ). Additionally, there was a less significant ( $P = 0.0190$ ) increase in SOD (superoxide dismutase) activity under EM exposure [77].

### 2.2.3. Problems in reproduction

In the town of Casavieja (Ávila, Spain) a telephony antenna was installed that had been in operation for about 5 years. Then some farmers began blaming the antenna for miscarriages in many pigs, 50–100 m from the antenna (on the outskirts of the town). Finally the topic became so bad that the town council decided to disassemble the antenna. It was removed in the spring 2005. From this moment onwards the problems stopped (C. Lumbreras personal communication).

A Greek study reports a progressive drop in the number of rodent births exposed to radiofrequencies. The mice exposed to 0.168  $\mu$ W/cm<sup>2</sup> become sterile after five generations, while those exposed to 1.053  $\mu$ W/cm<sup>2</sup> became sterile after only three generations [22].

In pregnant rats exposed to 27.12 MHz continuous waves at 100  $\mu$ W/cm<sup>2</sup> during different periods of pregnancy, half the pregnancies miscarried before the twentieth day of gestation, compared to only a 6% miscarriage rate in unexposed controls, and 38% of the viable foetuses had incomplete cranial ossification, compared to less than 6% of the controls. Findings included a considerable increase in the percentage of total reabsorptions (post-implantation losses consequent to RF radiation exposure in the first post-implantation stage). Reduced body weight in the exposed dams reflected a negative influence on their health. It seems that the irradiation time plays an important role in inducing specific effects consequent to radiofrequency radiation exposure [78]. There was also a change in the sex ratio, with more males born to rats that had been irradiated from the time of conception [2]. Moorhouse and Macdonald [79] find a substantial decline in female Water Vole numbers in the radio-collared population, apparently resulting from a male skew in the sex ratios of offspring born to this population. Recruits to the *radio-tracked* population were skewed heavily in favour of males (43:13). This suggests that radio-collaring of females caused male-skewed sex ratios.

Mobile phone exposure may have negative effects on sperm motility characteristics and male fertility in rats [46]. Other studies find a decrease of fertility, increase of deaths after birth and dystrophic changes in their reproductive organs [11]. Intermittent exposure showed a stronger effect than continuous exposure [4]. Brief, intermittent exposure to low-frequency EM fields during the critical prenatal period for neurobehavioral sex differentiation can demasculinize male scent marking behavior and increase accessory sex organ weights in adulthood [80].

In humans, magnetic field exposures above 2.0 mG were positively associated with miscarriage risk [81]. Exposure of pregnant women to mobile phone significantly increased foetal and neonatal heart rate, and significantly decreased the cardiac output [82].

#### 2.2.4. Nervous system

Microwaves may affect the blood brain barrier which lets toxic substances pass through from the blood to the brain [83]. Adang et al. [84] examined the effect of microwave exposure to a GSM-like frequency of 970 MHz pulsed waves on the memory in rats by means of an object recognition task. The rats that have been exposed for 2 months show normal exploratory behavior. The animals that have been exposed for 15 months show derogatory behavior. They do not make the distinction between a familiar and an unfamiliar object. In the area that received radiation directly from “Location Skrunda Radio Station” (Latvia), exposed children had less developed memory and attention, their reaction time was slower and neuromuscular apparatus endurance was decreased [85]. Exposure to cell phones prenatally and, to a lesser degree, postnatally was associated with behavioral difficulties such as emotional and hyperactivity problems around 7 years of age [86]. Electromagnetic radiation caused modification of sleep and alteration of cerebral electric response (EEG) [87–89]. Microwave radiation from phone masts may cause aggressiveness in people and animals (personal observation).

#### 2.3. Effects on amphibians

Disappearance of amphibians and other organisms is part of the global biodiversity crisis. An associated phenomenon is the appearance of large numbers of deformed amphibians. The problem has become more prevalent, with deformity rates up to 25% in some populations, which is significantly higher than previous decades [90]. Balmori [91] proposed that electromagnetic pollution (in the microwave and radiofrequency range) is a possible cause for deformations and decline of some wild amphibian populations.

Two species of amphibians were exposed to magnetic fields at various stages of development. A brief treatment of early amphibian embryos produced several types of abnormalities [92]. Exposure to a pulsed electromagnetic field produced abnormal limb regeneration in adult Newts [93]. Frog tadpoles (*Rana temporaria*) developed under electro-

magnetic field (50 Hz, 260 A/m) have increased mortality. Exposed tadpoles developed more slowly and less synchronously than control tadpoles and remain at the early stages for longer. Tadpoles developed allergies and EMF caused changes in blood counts [94].

In a current study exposing eggs and tadpoles ( $n=70$ ) of common frog (*R. temporaria*) for two months, from the phase of eggs until an advanced phase of tadpole, to four telephone base stations located 140 m away: with GSM system 948.0–959.8 MHz; DCS system: 1830.2–1854.8; 1855.2–1879.8 MHz. and UMTS system: 1905–1910; 1950–1965; 2140–2155 MHz. (electric field intensity: 1.847–2.254 V/m). A low coordination of movements, an asynchronous growth, with big and small tadpoles, and a high mortality (90%) was observed. The control group ( $n=70$ ), under the same conditions but inside a Faraday cage (metallic shielding component: EMC-reinforcement fabrics 97442 Marburg Technik), the coordination of movements was normal, the development was synchronously and the mortality rate was only 4.2% [95].

#### 2.4. Effects on insects

The microwaves may affect the insects. Insects are the basis and key species of ecosystems and they are especially sensitive to electromagnetic radiation that poses a threat to nature [96].

Carpenter and Livstone [97] irradiated pupae of *Tenebrio molitor* with 10 GHz microwaves at 80 mW for 20–30 min and 20 mW for 120 min obtained a rise in the proportion of insects with abnormalities or dead. In another study exposing fruit flies (*Drosophila melanogaster*) to mobile phone radiation, elevated stress protein levels (Hsp70) was obtained, which usually means that cells are exposed to adverse environmental conditions (‘non-thermal shock’) [98]. Panagopoulos et al. [99] exposed fruit flies (*D. melanogaster*) to radiation from a mobile phone (900 MHz) during the 2–5 first days of adulthood. The reproductive capacity of the species reduced by 50–60% in modulated radiation conditions (emission while talking on the phone) and 15–20% with radiation nomodulated (with the phone silent). The results of this study indicate that this radiation affects the gonadal development of insects in an athermal way. The authors concluded that radio frequencies, specifically GSM, are highly bioactive and provoke significant changes in physiological functions of living organisms. Panagopoulos et al. [100] compare the biological activity between the two systems GSM 900 MHz and DCS 1800 MHz in the reproductive capacity of fruit flies. Both types of radiation were found to decrease significantly and non-thermally the insect’s reproductive capacity, but GSM 900 MHz seems to be even more bioactive than DCS 1800 MHz. The difference seems to be dependent mostly on field intensity and less on carrier frequency.

A study in South Africa finds a strong correlation between decrease in ant and beetle diversity with the



electromagnetic radiation exposure (D. MacFadyen, personal communication.). A decrease of insects and arachnids near base stations was detected and corroborated by engineers and antenna's maintenance staff [101]. In houses near antennas an absence of flies, even in summer, was found.

In a recent study carried out with bees in Germany, only a few bees irradiated with DECT radiation returned to the beehive and they needed more time. The honeycomb weight was lower in irradiated bees [102]. In recent years a "colony collapse disorder" is occurring that some authors relate with pesticides and with increasing electromagnetic pollution [96].

The disappearance of insects could have an influence on bird's weakening caused by a lack of food, especially at the first stages in a young bird's life.

### 2.5. Effects on trees and plants

The microwaves may affect vegetables. In the area that received radiation directly from "Location Skrunda Radio Station" (Latvia), pines (*Pinus sylvestris*) experienced a lower growth radio. This did not occur beyond the area of impact of electromagnetic waves. A statistically significant negative correlation between increase tree growth and intensity of electromagnetic field was found, and was confirmed that the beginning of this growth decline coincided in time with the start of radar emissions. Authors evaluated other possible environmental factors which might have intervened, but none had noticeable effects [103]. In another study investigating cell ultrastructure of pine needles irradiated by the same radar, there was an increase of resin production, and was interpreted as an effect of stress caused by radiation, which would explain the aging and declining growth and viability of trees subjected to pulsed microwaves. They also found a low germination of seeds of pine trees more exposed [104]. The effects of Latvian radar was also felt by aquatic plants. *Spirodela polyrrhiza* exposed to a power density between 0.1 and 1.8  $\mu\text{W}/\text{cm}^2$  had lower longevity, problems in reproduction and morphological and developmental abnormalities compared with a control group who grew up far from the radar [105].

Chlorophylls were quantitatively studied in leaves of black locust (*Robinia pseudoacacia* L.) seedlings exposed to high frequency electromagnetic fields of 400 MHz. It was revealed that the ratio of the two main types of chlorophyll was decreasing logarithmically to the increase of daily exposure time [106].

Exposed tomato plants (*Lycopersicon esculentum*) to low level (900 MHz, 5 V/m) electromagnetic fields for a short period (10 min) measured changes in abundance of three specific mRNA after exposure, strongly suggesting that they are the direct consequence of application of radio-frequency fields and their similarities to wound responses suggests that this radiation is perceived by plants as an injurious stimulus [107]. Non-thermal exposure to radiofrequency fields

induced oxidative stress in duckweed (*Lemna minor*) as well as unespecific stress responses, especially of antioxidative enzymes [108].

For some years progressive deterioration of trees near phone masts have been observed in Valladolid (Spain). Trees located inside the main lobe (beam), look sad and feeble, possibly slow growth and a high susceptibility to illnesses and plagues. In places we have measured higher electric field intensity levels of radiation ( $>2 \text{ V/m}$ ) the trees show a more notable deterioration [109]. The tops of trees are dried up where the main beams are directed to, and they seem to be most vulnerable if they have their roots close to water. The trees don't grow above the height of the other ones and, those that stand out far above, have dried tops (Hargreaves, personal communication and personal observation). White and black poplars (*Populus sp.*) and willows (*Salix sp.*) are more sensitive. There may be a special sensitivity of this family exists or it could be due to their ecological characteristics forcing them to live near water, and thus electric conductivity. Other species as *Platanus sp.* and *Lygustrum japonicum*, are more resistant (personal observation). Schorpp [110] presents abundant pictures and explanations of what happens to irradiated trees.

### 3. Conclusions

This literature review shows that pulsed telephony microwave radiation can produce effects especially on nervous, cardiovascular, immune and reproductive systems [111]:

- Damage to the nervous system by altering electroencephalogram, changes in neural response or changes of the blood-brain barrier.
- Disruption of circadian rhythms (sleep-wake) by interfering with the pineal gland and hormonal imbalances.
- Changes in heart rate and blood pressure.
- Impairment of health and immunity towards pathogens, weakness, exhaustion, deterioration of plumage and growth problems.
- Problems in building the nest or impaired fertility, number of eggs, embryonic development, hatching percentage and survival of chickens.
- Genetic and developmental problems: problems of locomotion, partial albinism and melanism or promotion of tumors.

In the light of current knowledge there is enough evidence of serious effects from this technology to wildlife. For this reason precautionary measures should be developed, alongside environmental impact assessments prior to installation, and a ban on installation of phone masts in protected natural areas and in places where endangered species are present. Surveys should take place to objectively assess the severity of effects.

## Acknowledgment

The author is grateful to Denise Ward and Örjan Hallberg.

## References

- [1] J.M.R. Delgado, Biological effects of extremely low frequency electromagnetic fields, *J. Bioelectr.* 4 (1985) 75–91.
- [2] A. Firstenberg, *Microwaving Our Planet: The Environmental Impact of the Wireless Revolution*, 11210, Cellular Phone Taskforce, Brooklyn, NY, 1997.
- [3] A.L. Galeev, The effects of microwave radiation from mobile telephones on humans and animals, *Neurosci. Behav. Physiol.* 30 (2000) 187–194.
- [4] I. Belyaev, Non-thermal biological effects of microwaves, *Microw. Rev.* 11 (2005) 13–29, <http://www.mwr.medianis.net/pdf/Vol11No2-03-IBelyaev.pdf>.
- [5] H.P. Hutter, H. Moshhammer, P. Wallner, M. Kundi, Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations, *Occup. Environ. Med.* 63 (2006) 307–313.
- [6] T. Haumann, U. Munzenberg, W. Maes, P. Sierck, HF-radiation levels of GSM cellular phone towers in residential areas, in: 2nd International Workshop on Biological effects of EMFS, Rhodes, Greece, 2002.
- [7] W.R. Adey, Tissue interactions with non-ionizing electromagnetic fields, *Physiol. Rev.* 61 (1981) 435–514.
- [8] G.J. Hyland, Physics and biology of mobile telephony, *Lancet* 356 (2000) 1–8.
- [9] H. Lai, Biological effects of radiofrequency electromagnetic field, in: *Encyclopaedia of Biomaterials and Biomedical Engineering*, 2005, doi:10.1081/E-EBBE-120041846, pp. 1–8.
- [10] S. Kemerov, M. Marinkev, D. Getova, Effects of low-intensity electromagnetic fields on behavioral activity of rats, *Folia Med.* 41 (1999) 75–80.
- [11] N. Nikolaevich, A. Igorevna, and G. Vasil, Influence of high-frequency electromagnetic radiation at non-thermal intensities on the human body (A review of work by Russian and Ukrainian researchers), No place to hide, 3 (Supplement), 2001.
- [12] W.R. Adey, Bioeffects of mobile communications fields: possible mechanisms for cumulative dose. in: N. Kuster, Q. Balzano, J.C. Lin, (Eds.), *Mobile communications safety*, New York: Chapman & Hall, 1997, pp. 95–131.
- [13] A. Úbeda, M.A. Trillo, L. Chacón, M.J. Blanco, J. Leal, Chick embryo development can be irreversibly altered by early exposure to weak extremely-low-frequency magnetic fields, *Bioelectromagnetics* 15 (1994) 385–398.
- [14] I.U.G. Grigoriev, Role of modulation in biological effects of electromagnetic radiation, *Radiats. Biol. Radioecol.* 36 (1996) 659–670.
- [15] G.J. Hyland, The physiological and environmental effects of non-ionising electromagnetic radiation, Working document for the STOA Panel, European Parliament, Directorate General for Research, 2001.
- [16] R. Santini, J.M. Santini, P. danze, M. Leruz, M. Seigne, Enquête sur la santé de riverains de stations relais: I. Incidences de la distance et du sexe, *Pathol. Biol.* 50 (2002) 369–373.
- [17] R. Santini, P. Santini, J.M. Le Ruz, M. Danze, M. Seigne, Survey study of people living in the vicinity of cellular phone base stations, *Electromagn. Biol. Med.* 22 (2003) 41–49.
- [18] R. Santini, P. Santini, J.M. Danze, P. Le Ruz, M. Seigne, Symptoms experienced by people in vicinity of base stations: II/Incidences of age, duration of exposure, location of subjects in relation to the antennas and other electromagnetic factors, *Pathol. Biol.* 51 (2003) 412–415.
- [19] E.A. Navarro, J. Segura, M. Portolés, C. Gómez Perretta, The microwave syndrome: a preliminary study in Spain, *Electromagn. Biol. Med.* 22 (2003) 161–169.
- [20] G. Oberfeld, E. Navarro, M. Portoles, C. Maestu, C. Gomez-Perretta, The microwave syndrome—further aspects of a Spanish study, in: *EBEA Congres Kos, Greece*, 2004.
- [21] G. Abdel-Rassoul, M.A. Salem, A. Michael, F. Farahat, M. El-Batanouny, E. Salem, Neurobehavioral effects among inhabitants around mobile phone base stations, *Neurotoxicology* 28 (2007) 434–440.
- [22] I.N. Magras, T.D. Xenos, Radiation-induced changes in the prenatal development of mice, *Bioelectromagnetics* 18 (1997) 455–461.
- [23] A. Balmori, Possible effects of electromagnetic fields from phone masts on a population of white stork (*Ciconia ciconia*), *Electromagn. Biol. Med.* 24 (2005) 109–119.
- [24] J. Everaert, D. Bauwens, A possible effect of electromagnetic radiation from mobile phone base stations on the number of breeding House Sparrows (*Passer domesticus*), *Electromagn. Biol. Med.* 26 (2007) 63–72.
- [25] A. Balmori, Ö. Hallberg, The urban decline of the house sparrow (*Passer domesticus*): a possible link with electromagnetic radiation, *Electromagn. Biol. Med.* 26 (2007) 141–151.
- [26] M.J. Raven, D.G. Noble, S.R. Baillie, The breeding bird survey (2002), BTO Research Report 334, British Trust for Ornithology, Thetford, 2003.
- [27] J.D. Summers-Smith, The decline of the house sparrow: a review, *Brit. Birds* 96 (2003) 439–446.
- [28] J. De Laet, Ligue Royale Belge pour la Protection des Oiseaux avec l'Université de Gand, 2004, <<http://www.protectiondesoiseaux.be/content/view/801/74/>> (Accessed on May 20, 2008).
- [29] A. Prowse, The urban decline of the house sparrow, *Brit. Birds* 95 (2002) 143–146.
- [30] A. Balmori, Aves y telefonía móvil. Resultados preliminares de los efectos de las ondas electromagnéticas sobre la fauna urbana, *El ecologista* 36 (2003) 40–42.
- [31] C.K. Chou, A.W. Guy, L.L. Kunz, R.B. Johnson, J.J. Crowley, J.H. Krupp, Long-term, low-level microwave irradiation of rats, *Bioelectromagnetics* 13 (1992) 469–496.
- [32] E.T. Novoselova, E.E. Fesenko, Stimulation of production of tumour necrosis factor by murine macrophages when exposed in vivo and in vitro to weak electromagnetic waves in the centimeter range, *Biofizika* 43 (1998) 1132–1133.
- [33] K.J. Fernie, D.M. Bird, Evidence of oxidative stress in American kestrels exposed to electromagnetic fields, *Environ. Res. A* 86 (2001) 198–207.
- [34] R.C. Beasond, P. Semm, Responses of neurons to an amplitude modulated microwave stimulus, *Neurosci. Lett.* 33 (2002) 175–178.
- [35] K.J. Fernie, D.M. Bird, R.D. Dawson, P.C. Lague, Effects of electromagnetic fields on the reproductive success of American kestrels, *Physiol. Biochem. Zool.* 73 (2000) 60–65.
- [36] K.J. Fernie, N.J. Leonard, D.M. Bird, Behavior of free-ranging and captive American kestrels under electromagnetic fields, *J. Toxicol. Environ. Health, Part A* 59 (2000) 597–603.
- [37] K.J. Fernie, S.J. Reynolds, The effects of electromagnetic fields from power lines on avian reproductive biology and physiology: a review., *J. Toxicol. Environ. Health, Part B* 8 (2005) 127–140.
- [38] K.J. Fernie, D.M. Bird, Effects of electromagnetic fields on body mass and food-intake of American kestrels, *Condor* 101 (1999) 616–621.
- [39] S. Dasdag, M.A. Ketani, Z. Akdag, A.R. Ersay, I. Sar, Ö.C. Demirtas, M.S. Celik, Whole body microwave exposure emitted by cellular phones and testicular function of rats, *Urol. Res.* 27 (1999) 219–223.
- [40] M. Davoudi, C. Brössner, W. Kuber, Der Einfluss elektromagnetischer wellen auf die Spermienmotilität, *J. für Urol. Urogynäk.* 9 (2002) 18–22.
- [41] I. Fejes, Z. Za Vaczki, J. Szollosi, R.S. Kolosza, J. Daru, L. Kova Cs, L.A. Pa, Is there a relationship between cell phone use and semen quality? *Arch. Androl.* 51 (2005) 385–393.
- [42] P. Stefanis, A. Drakeley, R. Gazvani, D.I. Lewis-Jones, Growing concern over the safety of using mobile phones and male fertility, *Arch. Androl.* 52 (2006) 9–14.

- [43] O. Erogul, E. Oztas, I. Yildirim, T. Kir, E. Aydur, G. Komesli, H.C. Irkilata, M.K. Irmak, A.F. Peker, Effects of electromagnetic radiation from a cellular phone on human sperm motility: an in vitro study, *Arch. Med. Res.* 37 (2006) 840–843.
- [44] A. Agarwal, F. Deepinder, R.K. Sharma, G. Ranga, J. Li, Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study, *Fertil. Steril.* 89 (2008) 124–128.
- [45] A. Wdowiak, L. Wdowiak, H. Wiktor, Evaluation of the effect of using mobile phones on male fertility, *Ann. Agric. Environ. Med.* 14 (1) (2007) 169–172.
- [46] J.G. Yan, A.M. Gresti, T. Bruce, Y.H. Yan, A. Granlund, H.S. Matloub, Effects of cellular phone emissions on sperm motility in rats, *Fertil. Steril.* 88 (4) (2007) 957–964.
- [47] P.F. Doherty, T.C. Grubb, Effects of high-voltage power lines on birds breeding within the powerlines' electromagnetic fields, *Sialia* 18 (1996) 129–134.
- [48] V. Garaj-Vrhovac, D. Horvat, Z. Koren, The relationship between colony-forming ability, chromosome aberrations and incidence of micronuclei in V79 Chinese hamster cells exposed to microwave radiation, *Mutat. Res.* 263 (1991) 143–149.
- [49] H. Lai, N.P. Singh, Acute low-intensity microwave exposure increases DNA single-strand breaks in rat brain cells, *Bioelectromagnetics* 16 (1995) 207–210.
- [50] S. Balode, Assessment of radio-frequency electromagnetic radiation by the micronucleus test in bovine peripheral erythrocytes, *Sci. Total Environ.* 180 (1996) 81–85.
- [51] I. Belyaev, L. Hillert, E. Markova, R. Sarimov, L. Malmgren, B. Persson, M. Harms-Ringdahl, Microwaves of mobile phones affect human lymphocytes from normal and hypersensitive subjects dependent on frequency, in: 26th Annual Meeting of the Bioelectromagnetics (BEMS), Washington, USA, 2004.
- [52] G. Demisia, D. Vlastos, D.P. Matthopoulos, Effect of 910-MHz electromagnetic field on rat bone marrow, *Sci. World J.* 4 (2004) 48–54.
- [53] Reflex, 2004, <<http://www.verum-foundation.de/cgi-bin/content.cgi?id=euprojekte01>>.
- [54] E. Diem, C. Schwarz, F. Adlkofer, O. Jahn, H. Rudiger, Non-thermal DNA breakage by mobile-phone radiation (1800 MHz) in human fibroblasts and in transformed GFSH-R17 rat granulosa cells in vitro, *Mut. Res.* 583 (2005) 178–183.
- [55] A.G. Gandhi, P. Singh, Cytogenetic damage in mobile phone users: preliminary data, *Int. J. Hum. Genet.* 5 (2005) 259–265.
- [56] A. Di Carlo, N. Wite, F. Guo, P. Garrett, T. Litovitz, Chronic electromagnetic field exposure decreases HSP70 levels and lowers cytoprotection, *J. Cell. Biochem.* 84 (2002) 447–454.
- [57] D. Leszczynski, S. Joenväärä, J. Reivinen, R. Kuokka, Non-thermal activation of the hsp27/p38MAPK stress pathway by mobile phone radiation in human endothelial cells: molecular mechanism for cancer- and blood-brain barrier-related effects, *Differentiation* 70 (2002) 120–129.
- [58] E. Berman, L. Chacon, D. House, B.A. Koch, W.E. Koch, J. Leal, S. Lovtrup, E. Mantiply, A.H. Martin, G.I. Martucci, K.H. Mild, J.C. Monahan, M. Sandstrom, K. Shamsaifar, R. Tell, M.A. Trillo, A. Ubeda, P. Wagner, Development of chicken embryos in a pulsed magnetic field, *Bioelectromagnetics* 11 (1990) 169–187.
- [59] B.J. Youbicier-Simo, M. Bastide, Pathological effects induced by embryonic and postnatal exposure to EMFs radiation by cellular mobile phones, *Radiat. Protect.* 1 (1999) 218–223.
- [60] A. Ubeda, J. Leal, M.A. Trillo, M.A. Jimenez, J.M.R. Delgado, Pulse shape of magnetic fields influences chick embryogenesis, *J. Anat.* 137 (1983) 513–536.
- [61] J.M. Farrel, T.L. Litovitz, M. Penafiel, The effect of pulsed and sinusoidal magnetic fields on the morphology of developing chick embryos, *Bioelectromagnetics* 18 (1997) 431–438.
- [62] Ju.G. Grigoriew, Influence of the electromagnetic field of the mobile phones on chickens embryo, to the evaluation of the dangerousness after the criterion of this mortality, *J. Radiat. Biol.* 5 (2003) 541–544.
- [63] F. Batellier, I. Couty, D. Picard, J.P. Brillard, Effects of exposing chicken eggs to a cell phone in "call" position over the entire incubation period, *Theriogenology* 69 (2008) 737–745.
- [64] L. Veterány, A. Veterányová, J. Jedlicka, Effect of magnetic field on embryonic mortality, *Cesk. Fysiol.* 50 (2001) 141–143.
- [65] K.B. Hotary, K.R. Robinson, Evidence of a role for endogenous electrical fields in chick embryo development, *Development* 114 (1992) 985–996.
- [66] M. Mevissen, M. Häubler, Acceleration of mammary tumorigenesis by exposure of 7,12-dimethylbenz(a)anthracene-treated female rats in a 50-Hz, 100- $\mu$ T magnetic field: replication study, *J. Toxicol. Environ. Health, Part A* 53 (1998) 401–418.
- [67] D. Flipo, M. Fournier, C. Benquet, P. Roux, C. Le Boulaire, Increased apoptosis, changes in intracellular  $Ca^{2+}$ , and functional alterations in lymphocytes and macrophages after in vitro exposure to static magnetic field, *J. Toxicol. Environ. Health, Part A* 54 (1998) 63–76.
- [68] M. Fedrowitz, K. Kamino, W. Löscher, Significant differences in the effects of magnetic field exposure on 7,12 dimethylbenz (a)anthracene-induced mammary carcinogenesis in two sub-strains of Sprague-Dawley rats, *Cancer Res.* 64 (2004) 243–251.
- [69] A.H. Frey, S.R. Feld, Avoidance by rats of illumination with low power nonionizing electromagnetic energy, *J. Comp. Physiol. Psychol.* 89 (1975) 183–188.
- [70] M.A. Navakatikian, L.A. Tomashevskaya, Phasic behavioral and endocrine effects of microwaves of nonthermal intensity, in: D.O. Carpenter (Ed.), *Biological Effects of Electric and Magnetic Fields*, Academic Press, San Diego, CA, 1994.
- [71] D.D. Krstić, B.J. Đinđić, D.T. Sokolović, V.V. Marković, D.M. Petković, S.B. Radić, The results of experimental exposition of mice by mobile telephones, in: TELSIKS Conference, Serbia and Montenegro, Microw. Rev. (2005) 34–37.
- [72] I.U.G. Grigoriew, S.N. Luk'ianova, V.P. Makarov, V.V. Ryskov, N.V. Moiseeva, Motor activity of rabbits in conditions of chronic low-intensity pulse microwave irradiation, *Radiat. Biol. Radioecol.* 35 (1995) 29–35.
- [73] B. Nicholls, P.A. Racey, Bats avoid radar installations: Could electromagnetic fields deter bats from colliding with wind turbines? *PLOS One* 3 (2007) e297.
- [74] A. Balmori Murciélago rabudo-*Tadarida teniotis*, En: Enciclopedia Virtual de los Vertebrados Españoles, Carrascal, L.M., Salvador, A. (Eds.), Museo Nacional de Ciencias Naturales, Madrid, 2004c, <<http://www.vertebradosibericos.org/>>.
- [75] T.A. Marks, C.C. Ratke, W.O. English, Strain voltage and developmental, reproductive and other toxicology problems in dogs, cats and cows: a discussion, *Vet. Human Toxicol.* 37 (1995) 163–172.
- [76] W. Löscher, G. Käs, Conspicuous behavioural abnormalities in a dairy cow herd near a TV and radio transmitting antenna, *Pract. Vet. Surg.* 29 (1998) 437–444.
- [77] A. Yurekli, M. Ozkan, T. Kalkan, H. Saybasili, H. Tuncel, P. Atukeren, K. Gumustas, S. Seker, GSM Base Station Electromagnetic Radiation and Oxidative Stress in Rats, *Electromagn. Biol. Med.* 25 (2006) 177–188.
- [78] S. Tofani, G. Agnesod, P. Ossola, S. Ferrini, R. Bussi, Effects of continuous low-level exposure to radio-frequency radiation on intrauterine development in rats, *Health Phys.* 51 (1986) 489–499.
- [79] T.P. Moorhouse, D.W. Macdonald, Indirect negative impacts of radio-collaring: sex ratio variation in water voles, *J. Appl. Ecol.* 42 (2005) 91.
- [80] R.F. McGivern, R.Z. Sokol, W.R. Adey, Prenatal exposure to a low-frequency electromagnetic field demasculinizes adult scent marking behavior and increases accessory sex organ weights in rats, *Teratology* 41 (1990) 1–8.
- [81] G.M. Lee, R.R. Neutra, L. Hristova, M. Yost, R.A. Hiatt, A Nested Case-Control Study of Residential and Personal Magnetic Field Measures and Miscarriages, *Epidemiology* 13 (2002) 21–31.

- [82] A.Y. Rezk, K. Abdulqawi, R.M. Mustafa, T.M. Abo El-Azm, H. Al-Inany, Fetal and neonatal responses following maternal exposure to mobile phones, *Saudi Med. J.* 29 (2008) 218–223.
- [83] L.G. Salford, A.E. Brun, J.L. Eberhardt, L. Malmgren, B.R. Persson, Nerve cell damage in mammalian brain after exposure to microwaves from GSM mobile phones, *Environ. Health Perspect.* 111 (2003) 881–893.
- [84] D. Adang, B. Campo, A.V. Vorst, Has a 970 MHz Pulsed exposure an effect on the memory related behaviour of rats? in: The 9th European Conference on Wireless Technology, September 2006, 2006, pp.
- [85] A.A. Kolodynski, V.V. Kolodynska, Motor and psychological functions of school children living in the area of the Skrunda Radio Location Station in Latvia, *Sci. Total Environ.* 180 (1996) 87–93.
- [86] H.A. Divan, L. Kheifets, C. Obel, J. Olsen, Prenatal and postnatal exposure to cell phone use and behavioral problems in children, *Epidemiology* 19 (2008) 523–529.
- [87] K. Mann, J. Roschke, Effects of pulsed high-frequency electromagnetic fields on human sleep, *Neuropsychobiology* 33 (1996) 41–47.
- [88] A.V. Kramarenko, U. Tan, Effects of high-frequency electromagnetic fields on human EEG: a brain mapping study, *Int. J. Neurosci.* 113 (2003) 1007–1019.
- [89] A.A. Marino, E. Nilsen, C. Frilot, Nonlinear changes in brain electrical activity due to cell phone radiation, *Bioelectromagnetics* 24 (2003) 339–346.
- [90] A.R. Blaustein, P.T.J. Johnson, Explaining frog deformities, *Sci. Am.* 288 (2003) 60–65.
- [91] A. Balmori, The incidence of electromagnetic pollution on the amphibian decline: is this an important piece of the puzzle? *Toxicol. Environ. Chem.* 88 (2006) 287–299.
- [92] W.C. Levengood, A new teratogenic agent applied to amphibian embryos, *J. Embryol. Exp. Morphol.* 21 (1969) 23–31.
- [93] R.H. Landesman, W. Scott Douglas, Abnormal limb regeneration in adult newts exposed to a pulsed electromagnetic field, *Teratology* 42 (1990) 137–145.
- [94] N.M. Grefner, T.L. Yakovleva, I.K. Boreyshe, Effects of electromagnetic radiation on tadpole development in the common frog (*Rana temporaria* L.), *Russ. J. Ecol.* 29 (1998) 133–134.
- [95] A. Balmori, in preparation: Phone masts effects on common frog (*Rana temporaria*) tadpoles: An experiment in the city. See the video clips in: <http://www.hese-project.org/hese-uk/en/issues/nature.php?id=frogs>.
- [96] U. Warnke, Bienen, vögel und menschen, Die Zerstörung der Natur durch “Elektrosmog”. Kompetenzinitiative, 2007 46 pp.
- [97] R.L. Carpenter, E.M. Livstone, Evidence for nonthermal effects of microwave radiation: Abnormal development of irradiated insect pupae, *IEEE Trans. Microw. Theor. Tech.* 19 (1971) 173–178.
- [98] D. Weisbrot, H. Lin, L. Ye, M. Blank, R. Goodman, Effects of mobile phone radiation on reproduction and development in *Drosophila melanogaster*, *J. Cell. Biochem.* 89 (2003) 48–55.
- [99] D.J. Panagopoulos, A. Karabarbounis, L.H. Margaritis, Effect of GSM 900 MHz Mobile Phone Radiation on the Reproductive Capacity of *Drosophila melanogaster*, *Electromagn. Biol. Med.* 23 (2004) 29–43.
- [100] D.J. Panagopoulos, E.D. Chavdoula, A. Karabarbounis, L.H. Margaritis, Comparison of bioactivity between GSM 900 MHz and DCS 1800 MHz mobile telephony radiation, *Electromagn. Biol. Med.* 26 (2007) 33–44.
- [101] A. Balmori, Efectos de las radiaciones electromagnéticas de la telefonía móvil sobre los insectos, *Ecosistemas* (2006).
- [102] H. Stever, J. Kuhn, C. Otten, B. Wunder, W. Harst, Verhaltensänderung unter elektromagnetischer Exposition. Pilotstudie, Institut für mathematik. Arbeitsgruppe, Bildungsinformatik. Universität Koblenz-Landau, 2005.
- [103] V.G. Balodis, K. Brumelis, O. Kalvickis, D. Nikodemus, V.Z. y Tjarve, Does the Skrunda radio location station diminish the radial growth of pine trees? *Sci. Total Environ.* 180 (1996) 57–64.
- [104] T. Selga, M. Selga, Response of *Pinus Sylvestris* L. needles to electromagnetic fields. Cytological and ultrastructural aspects, *Sci. Total Environ.* 180 (1996) 65–73.
- [105] I. Magone, The effect of electromagnetic radiation from the Skrunda Radio Location Station on *Spirodela polyrhiza* (L.) Schleiden cultures, *Sci. Total Environ.* 180 (1996) 75–80.
- [106] D.D. Sandu, C. Goiceanu, A. Ispas, I. Creanga, S. Miclaus, D.E. Creanga, A preliminary study on ultra high frequency electromagnetic fields effect on black locust chlorophylls, *Acta Biol. Hung.* 56 (2005) 109–117.
- [107] D. Roux, A. Vian, S. Girard, P. Bonnet, F. Paladian, E. Davies, G. Ledoigt, High frequency (900 MHz) low amplitude ( $5 \text{ V m}^{-1}$ ) electromagnetic field: a genuine environmental stimulus that affects transcription, translation, calcium and energy charge in tomato, *Planta* 227 (2007) 883–891.
- [108] M. Tkalec, K. Malarik, B. Pevalek-Kozlina, Exposure to radiofrequency radiation induces oxidative stress in duckweed *Lemna minor* L., *Sci. Total Environ.* 388 (2007) 78–89.
- [109] A. Balmori, ¿Pueden afectar las microondas pulsadas emitidas por las antenas de telefonía a los árboles y otros vegetales? *Ecosistemas* (2004), <http://www.revistaecosistemas.net/articulo.asp?Id=29&Id.Categoria=1&tipo=otros.contenidos>.
- [110] V. Schorpp, 2007, <<http://www.puls-schlag.org/baumschaeden.htm#linden>>.
- [111] A. Balmori, Posibles efectos de las ondas electromagnéticas utilizadas en la telefonía inalámbrica sobre los seres vivos, *Ardeola* 51 (2004) 477–490.



## FM-radio and TV tower signals can cause spontaneous hand movements near moving RF reflector

Paavo Huttunen<sup>a,\*</sup>, Osmo Hänninen<sup>b</sup>, Risto Myllylä<sup>a</sup>

<sup>a</sup> *Laboratory of Optoelectronics and Measurement Technology, University of Oulu, P.O. Box 4500, 90014 Oulu, Finland*

<sup>b</sup> *Department of Physiology, University of Kuopio, P.O. Box 1627, 70211 Kuopio, Finland*

Received 2 January 2009; received in revised form 23 January 2009; accepted 30 January 2009

### Abstract

For testing human sensitivity to radio frequency (RF) standing waves a movable reflecting wall was constructed. Radio waves from the radio-TV tower reflected back and formed a standing wave near the reflector. When the reflector was moved, the position of the maximums of the standing waves changed and the electromagnetic intensity changed in the body of the standing test subject. The computer with an AD-converter registered the signals of the hand movement transducer and the RF-meter with 100 MHz dipole antennas. A total of 29 adults of different ages were tested. There were 9 persons whose hand movement graphs included features like the RF-meter. Six showed responses that did not correlate with the RF-meter. There were also 14 persons who did not react at all. Sensitive persons seem to react to crossing standing waves of the FM-radio or TV broadcasting signals.

© 2009 Elsevier Ireland Ltd. All rights reserved.

**Keywords:** Sensorimotor responses; Radio frequency standing waves

### 1. Introduction

Radio frequency radiation (RFR) has been studied intensively in the near GHz region. Subjective symptoms, sleeping problems and cognitive performance have been reported in subjects living near mobile phone base stations [1]. In the recent past, frequencies of FM-radio and television (TV) signals have been much less studied even though these frequencies cause biological and health effects, too. The whole body resonance frequency of an average man and thus the maximum absorption of RF energy occur at 70–80 MHz [2]. This is near the frequencies used in very high frequency (VHF) broadcasting. The head and limbs absorb much more energy than the torso at frequencies above body resonance [3]. Greatest absorption in the head region of man occurs at a frequency of about 375 MHz [4]. Absorption is stronger for wave propagation from head to toe than it is when the electric field is parallel to the long axis. The authors [4] believed that the enhanced absorption in the head region may make

head resonance significant in the study of behavioral effects, blood–brain barrier permeability, cataractogenesis, and other microwave bioeffects. Even increased health risks like cancer, especially melanoma incidence, near FM broadcasting and television transmitters have been reported [5,6].

Nerve impulses initiate muscle contraction by calcium ion release from the sarcoplasmic reticulum, which takes place when electric nerve signals reach the plasma membrane and T-tubules of muscle fibers [7]. Voltage dependent Ca-channels open. Acetylcholine esterase (AChE) breaks down the acetylcholine, and Na-channels close [7]. It has been reported that the number of Ca<sup>2+</sup> ions liberated from hen's frontal brain depends on the modulation frequency of the weak VHF radiation, with a maximum at a frequency of 16 Hz, while an unmodulated field causes no ion release [2,8]. Multiple RF power-density windows in calcium ion release from brain tissue have presented [9]. A significant decrease in AChE activity has been found in rats exposed to radio frequency radiation of 147 MHz and its sub-harmonics 73.5 and 36.75 MHz amplitude modulated at 16 and 76 Hz. A decrease in AChE activity was independent of carrier wave frequencies [10].

\* Corresponding author.

E-mail address: paavo.huttunen@elisanet.fi (P. Huttunen).

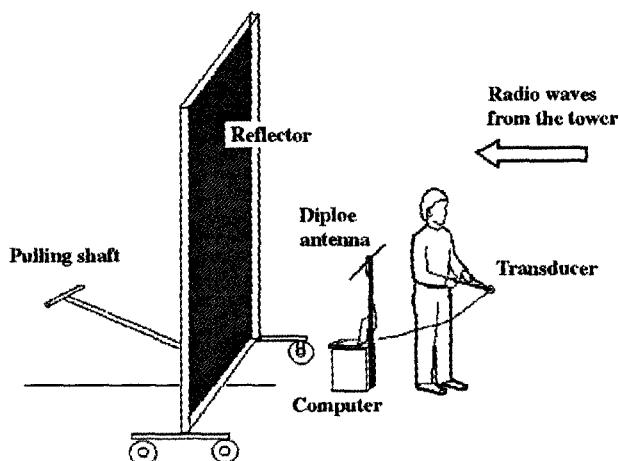


Fig. 1. Testing human radio wave sensitivity. Radio waves from the TV tower reflect back from the reflector and form a standing wave. When the reflector moves, the position of the maximums of the standing wave change, and the electromagnetic intensity changes in the body of the test subject. The computer with an AD-converter registers the signals of hand movement transducer and the RF-meter with the dipole antennas.

As there is previous evidence from human and animal studies that electromagnetic irradiation has effects in the brain, the aim of the present study was to find out, if the motor responses are generated in sensitive persons, when they move across a set of standing waves caused by radiation of a FM-radio and TV tower. The connection between the hand movements and the integrated intensity of electromagnetic field of FM-radio broadcasting were recorded.

## 2. Methods

The wavelength of a 100-MHz radio wave is 3 m. For testing human sensitivity to moving standing waves a movable reflecting wall with wooden frame 3 m height and 5 m wide was constructed (Fig. 1). Steel net of 20 mm × 20 mm mesh was used. Five horizontal net slices of 60 cm wide were bound together with steel wire forming a radio waves reflecting surface. The test place was 5 km from the FM-radio tower. The frame was placed in an open field perpendicular to the incoming wave. The test subject was standing back towards the frame, and he had the hand movement transducer in his hands. The RF-meter with horizontal dipole antenna was close behind him. When started, the frame was 2 m from his back and it was moved 20 m forth and back. The computer registered both signals. The method and the aim of the test were at first presented, in brief, to the test persons. All together 29 adult persons of different ages were tested. They were participants in a seminar relating to effects of electric fields, and thus they possibly do not represent a normal population.

The broadband (30–300 MHz) RF-meter and the hand movement transducer were constructed for this study by the authors. The signals were digitised by Pico high resolution

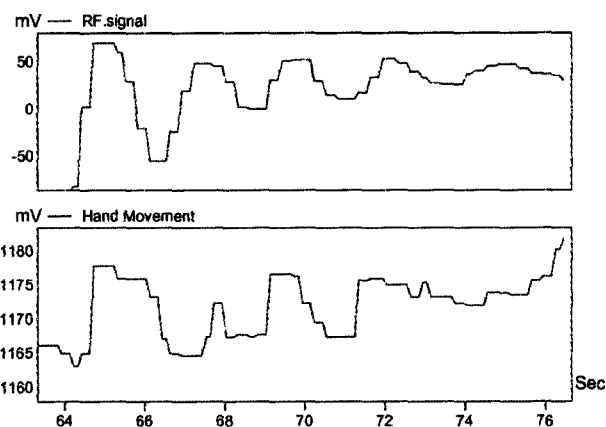


Fig. 2. Hand movements near the moving RF reflector. The standing waves moved slowly with the reflector. Intensity of the electric field was measured with the broadband RF-meter with horizontal dipole antennas. Variation of the field intensity is presented in the upper curve and the hand movements of the standing test person are in the lower curve.

data logger (ADC16). The radio frequency spectrum was measured using a spectrum analyser (GW instek GSP-827, 2.7 GHz) with 1.5 m horizontal dipole antennas. When measured, the antenna was fastened to a wooden frame 1 m from the ground.

## 3. Results and discussion

Results on the movable frame showed different hand movement reactions of the test subjects. There were 9 persons who reacted like the RF-meter (Fig. 2), 6 persons whose graphs, though obvious, showed no correlation to the RF-meter and 14 persons who did not react or showed only small noise like changes in their graphs (Table 1). Spectrum at the test place contains mainly the FM-radio broadcasting signals and four digital TV signals (Fig. 3). Most prominent (85 dB  $\mu$ V, approximately 50 mV/m) are the 6 horizontally polarized FM-radio signals (Fig. 4).

Resonances in body parts affects the power absorption. Theoretically, the optimal length of a thin antenna in radio-frequency reception is nearly half of the wavelength of the

Table 1

Reactions to standing waves of FM-radio signals. Classification of results of 29 tested persons. Test subject was standing and the radio wave reflector was moved behind him/her. The hand movement graphs were compared to the graphs of the broadband radio frequency (RF) meter.

Reactions to standing waves	9 persons	Hand movement graphs include features like graphs of RF-meter.
Possible reaction	6 persons	Changes in the graphs but no correlation to RF-meter.
No reaction	14 persons	Only small noise like changes in the graphs.



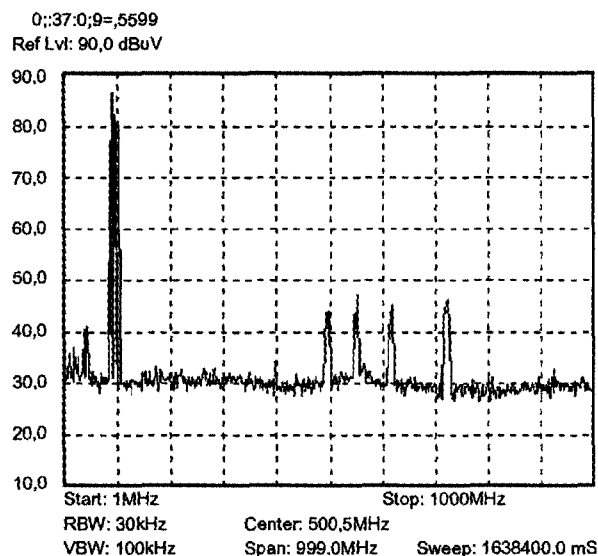


Fig. 3. Spectrum 1–1000 MHz at the test place. The highest peaks at the left are FM-radio broadcasting signals and the four lower peaks in the middle are the digital TV signals. Because the measurement was made with 1.5 m dipoles, signals near 100 MHz are more prominent because of antenna resonance.

incoming radio wave. The experimental maximum whole body resonance frequency is lower than the resonance frequency for an ideal half wave dipole antenna [11]. The whole body resonance length of a human at the frequencies of 80–108 MHz applied to FM broadcasting is about 1.1–1.5 m. Because in this experiment the test subjects were standing and the 100 MHz FM-radio signals and TV signals at higher frequencies are horizontally polarized, the absorption is obviously higher in the shoulder area. The distance between two maximums of the 100 MHz standing wave is 1.5 m. The half

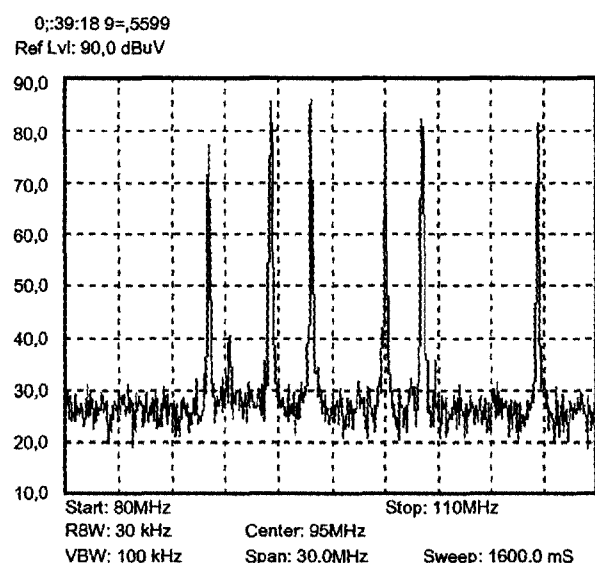


Fig. 4. Spectrum of the FM-radio broadcasting at the test place. Six channels were sending and the maximum electric field intensity was 85 dB  $\mu$ V.

waves of local digital TV signals (500–700 MHz) are only about 20–30 cm. This means that there can be many maximums of standing waves of TV signals in the body at the same time, even near the reflector.

The biggest variation in the local field intensity was caused by the FM broadcasting. There were 6 channels in the tower. Because of different wave lengths, the standing waves near the reflector are at the same phase and they amplify each other, but further away, the phases are mixed and so the amplitude of the summed standing waves is smaller.

With this experiment, we cannot exactly say where the reaction occurs, in limbs, muscles or in the head. It is possible that a change of intensity in standing radiowaves causes a small change in the nerve-muscle permeability of the nerve signal. The person feels it like a spontaneous muscle contraction. His hands are moving away and closer when the standing waves are passing. By some persons, the distance from hand to hand varied 0–60 cm. That means that some of muscles in arms and shoulders should react.

The spectrum contains many frequencies of electromagnetic radiation. The radiation is not only coming from the nearest tower, and it is impossible to clean the test area from other waves. This experiment was made at rural area, but even there, the private hand held telephone signals cause interferences to RF-instruments.

#### 4. Conclusions

Sensitive persons seem to react to crossing standing waves of the FM-radio or TV broadcasting signals. The reactions were apparently initiated by RFR near reflecting objects, but they became more random in very weak variations of total field intensity. In any case, individuals are different, and in natural situations many sources interfere with each other.

#### References

- [1] H.-P. Hutter, H. Moshhammer, P. Wallner, M. Kundi, Subjective symptoms, sleeping problems, and cognitive performance in subjects living near mobile phone base stations, *Occup. Environ. Med.* 63 (2006) 307–313.
- [2] R.V. Adey, Frequency and power windowing in tissue interactions with weak electromagnetic fields, *Proc. IEEE* 68 (1) (1980) 119–125.
- [3] R.G. Olsen, T.A. Griner, Specific absorption rate in models of man and monkey at 225 and 2,000 MHz, *Bioelectromagnetics* 8 (1987) 377–384.
- [4] M.J. Hagmann, O.P. Gandhi, J.A. D'andrea, I. Chatterjee, Head resonance: numerical solutions and experimental results, *IEEE Trans. Microw. Theory Tech.* MTT-27 (9) (1979) 809–813.
- [5] H.G. Dolk, P. Shaddik, Walls, et al., Cancer incidence near radio and television transmitters in Great Britain I and II, *Am.J. Epidemiol.* 145 (1997) 1–17.
- [6] Ö. Hallberg, O. Johansson, Melanoma incidence and frequency modulation (FM) broadcasting, *Arch. Environ. Health* 57 (1) (2002) 32–40.
- [7] B. Alberts, D. Bray, J. Lewis, M. Raff, K. Roberts, J.D. Watson (Eds.), *Molecular Biology of the Cell, The Nervous System*, Garland Publishing Inc., New York/London, 1983, pp. 1013–1098 (Chapter 18).